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BULLETIN

OF

THE JOHNS HOPKINS HOSPITAL.

Vol. IX.—No. 92.] BALTIMORE, NOVEMBER, 1898. [Price, 15 Cents.

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THE DIAGNOSIS OF THE CONDITION OF EACH KIDNEY BY INOCULATION OF THE SEPARATED SEDIMENTS INTO GUINEA-PIGS IN SUSPECTED RENAL TUBERCULOSIS.

By Edward Reynolds, M. D., *Boston, Mass.*

The direct inspection of the air-distended bladder which we owe to Kelly, with its sequelæ of easy exploration of the ureters, has already led to great advances towards an accurate knowledge of the urinary diseases of women, and at the present moment, when so much is opening up before us, any new step towards exactness of diagnosis seems worth reporting.

Little is yet known of the natural history of renal tuberculosis; indeed, it is for comparatively few years that we have known that tuberculosis can be primary in the kidney, and the great mass of the profession has not yet realized that this disease is often localized for many years in one kidney before invading the rest of the urinary tract; but in the last three years we have been accumulating a considerable amount of clinical evidence in support of these views, namely, that though renal tuberculosis does in the end kill when untreated, it is often nevertheless so strictly localized in one kidney that the patient may be restored to perfect health after this is removed by nephrectomy. Among the many cases of this nature which we now have may be cited a remarkable one by Vineburg* and three by Kelly.† I am myself able to add three unpublished cases, two of my own and one very remarkable case which I saw in consultation after a nephrectomy by another surgeon. Those who have ever seen how these patients are transformed in nutrition and general health, by the removal of the offending kidney, will be slow to listen to arguments against the operation for cases in which the disease is unilateral, but it is of course justifiable only in such cases, and our success must therefore rest on our power of diagnosing the disease while it is limited to one kidney, and thus confining the operation to cases which are capable of cure by nephrectomy. For this purpose we must not only be able to establish the diagnosis of renal tuberculosis, in advance of the constitutional breakdown of the patient, but must also be able to satisfy ourselves with some positiveness of the health of the other kidney; and striking testimony to the advances which have been made in this subject during the last few years is to be obtained by inspection of the most recent text-books on medical diagnosis. Thus one, which shall be nameless, in the course of three pages devoted to renal tuberculosis, gives under diagnosis only these three clues: the presence in the sediment of the urine of little, yellow, cheesy masses of degenerated tuberculous material; the presence of pus and other signs of chronic pyelitis from no assignable cause other than tubercle, and, lastly, the presence of tuberculosis in other organs. Even the latest edition of Osler, in which the section on this subject

*Medical Record, Feb. 6th, 1898.

†Johns Hopkins Hospital Bulletin, Feb.-Mar., 1896.

has been considerably rewritten and evidently brought up to date, may be summarized as saying that there is but little chance of making a diagnosis unless we are put upon the track by finding tuberculosis in other organs, though this acute writer does mention the special methods of examination employed by gynecologists and the hope for the future which they afford.

It is apparent that these medical descriptions refer to what we from a surgical standpoint should call advanced disease. Our surgical specialty has already led us to the possibility of establishing the diagnosis at a very much earlier stage than this.

Though such a diagnosis can be made only by physical examination, a suspicion of tubercular or other renal disease will often be excited by the symptomatology, and this must therefore be briefly reviewed. The early symptoms of the disease will vary greatly both in character and in intensity in different cases, and from time to time in the same case, but will consist typically of pain and tenderness over the abdomen on the affected side, frequency of urination, and sometimes hæmaturia. These symptoms are, however, equally characteristic of simple pyelitis, renal calculus and new growth in the pelvis of the kidney. There are, indeed, no differences of kind in the symptomatology of these diseases in their early stages, though there are differences in the degree in which the several symptoms are likely to be present.

All these diseases are characterized by dull pains over the kidney and along the course of the ureter on the affected side. The pain is often bearing-down in character, and therefore a uterine origin is usually assigned to it; is usually increased by standing; and is always more or less associated with frequency of micturition, occurring at night as well as in the day-time. In all of them the call to micturate is a very urgent one; the pain on micturition is usually referred to the meatus; and all are liable to more or less tenesmus at the end of micturition. In all the pain may be increased by jarring or jolting (as in driving over rough roads).

This whole group of symptoms is, however, in reality symptomatic of the amount of inflammation present, and therefore varies with this subsidiary feature of the case. They are often though not always more marked in renal stone than in the other diseases.

All the diseases enumerated may be accompanied by hæmaturia, but this is rare in simple pyelitis; it is more likely to be profuse in tuberculosis or a new growth than in stone.

In all, the patients are almost equally liable to attacks of mild renal colic, due usually to inflammatory obstruction in the ureter. They differ from the pains excited by the passage of a calculus in being less severe and not followed by the appearance of the stone. These attacks are perhaps less common in new growths than in the other diseases under consideration. Not infrequently, at intervals in the course of these chronic renal diseases (and especially in tuberculosis and simple pyelitis), the inflammatory symptoms will be found to be most marked on the sound side. This is probably because each exacerbation of the disease in the affected side leads to an increased elimination of toxic materials from the functionally more active kidney; and this excites a transitory and somewhat acute inflammation in the mucous membrane of the urinary apparatus on the sound side.

This transposition of symptoms I have seen so frequently (I may say almost constantly) that I am sure it must always be guarded against. The side on which the patient tells us that the symptoms were first noticed is usually the diseased side.

The inquiry into the history should be followed by palpation, both abdominal and bimanual. In all these lesions we find, on palpation, a tenderness which may extend from top to bottom of the urinary tract on the affected, and even on both sides, but which is usually most marked at one or more of three points: namely, over the kidney; at the spot where the ureter crosses the brim of the pelvis, in which case it is often limited to a spot the size of the finger-tip, midway between the umbilicus and the anterior superior spine of the ilium (McBurney's point or its fellow); and, finally, over the vesical end of the ureter at the side of the cervix, which examination may even detect an enlargement or induration of the ureter. These tender points are again symptomatic of the amount of inflammation present, and therefore usually vary with the amount of pain.

A study of the history and the results of the gynecological examination usually enables us, then, to suspect, and sometimes permits us to postulate, a diagnosis of some renal disease of a surgical nature, but it does not enable us to say what, nor always on which side it is. The special examination now steps in and the real diagnosis begins here.

If a visual examination of the bladder shows that the vesical mucous membrane is substantially normal except in the interureteral region, and that a strongly localized inflammation is present there, the source of that inflammation is probably to be found in the passage of a vicious urine from one or the other ureter. If one ureteral orifice is abnormal in appearance, this probably marks the diseased side. The next and most important step is the catheterization of the ureters and a microscopical and chemical examination of the urine secreted by each kidney. But here the results must be interpreted with the greatest care, as recent advances have made it certain that most of our past opinions on the results of urinalysis must be revised in the light of the new knowledge. This is especially true of renal tuberculosis; the large amounts of degenerated pus and bits of necrotic material which are commonly described as characteristic of the disease being, in fact, found only in locally advanced cases, while the urinary signs of early tuberculosis are usually limited to the detection of pus and the bacilli by the microscope.

In the more advanced of the class of cases which are still quite operable we are indeed almost sure to find more or less degenerated pus, but the amount of it varies greatly from time to time, and is not infrequently insignificant. The detection of tubercle bacilli in the sediment of the urine is of some positive value (it is absolute if its confusion with the smegma bacillus is sufficiently carefully excluded; and it should therefore be absolute in the sediment of the urine obtained by ureteral catheterization). Its absence is of no diagnostic value whatever; *e. g.* I have had a highly trained expert make repeated negative reports on the urine from a

bladder in which tubercular ulcerations were actually visible and in which a subsequent report was positive.

It is probable that the discharge of the bacilli with the urine is not uniform, and no man can expect to search a sediment so thoroughly as to detect the bacillus with certainty if only a few are present; but it is held that if a fresh sediment is injected into the peritoneum of a guinea-pig, the presence of only one or two bacilli will be enough to cause infection in this very sensitive animal. The generally accepted opinion that this is the most delicate test for tuberculosis known, and the great surgical importance of using the most delicate test possible for the determination of the condition of each kidney separately in suspected renal tuberculosis, has then been my reason for injecting the sediments obtained from the kidneys by ureteral catheterization into separate guinea-pigs in each of three cases of suspected renal tuerculosis.

If this test is to be of real surgical value two points must be determined with regard to it: 1. Will it give us positive evidence in the early stages of the disease? 2. How absolute is the negative evidence obtained by the negative results from the other kidney in the same case? But neither of these questions can be answered by anything but an extended experience.

One case was positive as regards one kidney only, both the others were wholly negative, though each woman showed evidence of surgical disease in one of her kidneys. I have put off the publication of this report for several months in the hope of reporting the ultimate results in the equally important negative cases, but as both patients are still deferring operation* I am publishing the one positive result in the hope of inducing others to try this very delicate test, and also because the individual case is of itself of much interest from the slight development of the disease in the kidney which was removed, and from the very satisfactory improvement in the patient's condition since operation, which certainly so far supports the negative result obtained from the examination of her remaining kidney.

On March 21, 1898, I saw, with Dr. Percy C. Proctor, of Gloucester, Mass., Mrs. P., thirty-one years old, ten years married, multipara, of tuberculous family history, but with good personal history until the beginning of the present illness.

Eight years ago, after suffering for some months from backache and bearing-down pain, she suddenly began to pass bloody urine, which gradually returned to the normal after a duration of some weeks. Though partially relieved of her backache after the attack, she has been a semi-invalid ever since. Four years later, in October of 1894, she had a second, similar attack, and on the 17th of February, 1898, a third attack, the hæmaturia beginning with equally little warning. The pain has always been relieved during the attacks, but has always returned after their cessation, and has never been affected by the act of urination. During these attacks she has passed urine about every half hour during the day, but only once or not at all during the night. In the intervals between the attacks she has had no frequency or other abnormalities of urination, *i. e.* the symptomatology was indistinctive.

On palpation no tenderness could be detected. On examination under ether her genital organs were essentially normal, and a careful visual inspection of the bladder showed no abnormality whatsoever, except that a stream of thin blood trickled steadily downward from the right ureteral orifice, while from the left spouted intermittently a normal looking urine.

The ureters were catheterized and the specimens were submitted to Dr. J. B. Ogden, Assistant in Chemistry in the Harvard Medical School, whose reports of the examination of the urine and of the inoculations which he made at my request are here appended:

"On March 22, 1898, two specimens of urine from the right and left ureters—case Mrs. P.—were submitted to me by Dr. Edw. Reynolds, for examination.

Urine from Right Ureter.—Amount received 10 cc.; color, bloody; reaction, alkaline; specific gravity could not be taken as quantity of urine was not sufficient; urea, 1.01 per cent.; albumin, between $\frac{1}{4}$ and $\frac{1}{2}$ of 1 per cent. The sediment, which was abundant, consisted chiefly of normal blood. After the blood had been destroyed by means of distilled water, which had been acidulated with acetic acid numerous leucocytes, a few small round and caudate cells (probably ureteral) and rarely a brown granular cast were found. No crystalline elements detected.

Urine from Left Ureter.—Amount received 3 cc.; color, pale, slightly turbid; reaction, acid; specific gravity could not be taken; urea, 0.95 per cent.; albumin, a trace. The sediment contained frequent normal and abnormal blood globules and few leucocytes, many medium and small round cells, and numerous small caudate cells as from the ureter. An occasional granular and brown granular cast, and uric acid crystal.

The clinical examination of these specimens did not reveal much toward deciding as to the most probable cause of the clinical symptoms. The uric acid crystals suggested a possible cause of the trouble, but as it was several hours after the urines had been collected before a microscopical examination could be made, they were probably secondary (formed and deposited after the collection of the urine).

The considerable quantity of normal blood in the urine from the *right* ureter was apparently of traumatic origin, and was the probable cause of the alkalinity of the urine. The presence of numerous leucocytes in the sediment led to the question: Are there more leucocytes than can be accounted for by the amount of blood present? This I was unable to fully decide, although they appeared to be present in somewhat larger numbers than would be expected in that quantity of blood.

The tubular disturbance shown by the presence of casts, although slight, appeared to be more marked in the left kidney than in the right.

Since the question of a tuberculosis of the urinary tract had been raised as a possible cause of the symptoms in this case, it was considered advisable to make as thorough and complete an examination of the urine for tubercle bacilli as was possible, and at the same time, if a tuberculosis existed, to determine whether one or both kidneys were diseased. Accordingly the sediments of both specimens were washed twice, by decantation, with distilled water, in order to free them from albumin and other soluble urinary constituents, a centrifuge being used to settle the sediments after each addition of water. Each sediment was then divided into two portions; one portion was injected into a guinea-pig, and the other was reserved for microscopical examination for tubercle bacilli.

* While the paper was in press both negative cases came to operation, and both proved to have non-tubercular disease. Both will be published in detail later.

The injections into the guinea-pigs were made as follows: The barrel and needle of a small Koch syringe were thoroughly sterilized by dry heat; the abdominal wall of a guinea-pig was thoroughly cleansed and then ½ cc. of the sediment of the urine from the *right* ureter was injected into the abdominal cavity. The barrel and needle of the syringe were cleansed and again sterilized. The abdominal wall of another guinea-pig was cleansed, after which ½ cc. of the sediment of the urine from the *left* ureter was injected into the abdominal cavity.

The pigs, following the injections, showed only slight disturbance, from which they recovered in a few days, and were apparently quite well for the eight weeks they were under observation. An examination of the pigs between the fifth and sixth weeks showed that the one which had been inoculated with the sediment of the urine from the right kidney had, in both groins, enlarged glands, which were hard and quite nodular. The one injected with the sediment of the urine from the left kidney showed no enlarged glands and was apparently in a healthy condition. Both animals were then placed under the care of Dr. W. F. Whitney.

On the same day that the inoculations were made the portion of sediment which had been reserved for microscopical examination was centrifugalized, and the sediment placed on cover-glasses was carefully dried, stained and examined. No tubercle bacilli could be found in the limited number of preparations at hand. The amount of sediment furnished by these small specimens was originally comparatively slight, and since some of the sediment had been used for the inoculation experiments, too little remained for as *thorough* a microscopical examination as is often necessary for the detection of tubercle bacilli in the urine."

The patient was kept absolutely in bed on a non-stimulating diet, but the hæmaturia continued until the latter part of April, when the bleeding ceased and the pain in the back returned. As I thought it advisable to give her some weeks in which to regain condition, the pigs were left undisturbed till some weeks later, when I received the following letters from Dr. W. F. Whitney, curator of the Warren Anatomical Museum and Pathologist to the Massachusetts General Hospital.

"May 26, 1898. The guinea-pig inoculated with the urine from the right kidney of Mrs. P. was killed to day, and showed cheesy abscesses in the glands of both groins, in the pus from which a few scattered tubercle bacilli were found. There were also characteristic miliary cheesy nodules in the spleen, as well as a few scattered areas in the liver. The condition is perfectly characteristic of inoculated tuberculosis."

"June 13, 1898. The guinea-pig inoculated with the urine from the left kidney of Mrs. P. on March 22, 1898, was killed June 9, 1898, and found to be perfectly normal."

As I always think it a pity to disturb the ureter of the sound side by catheterization immediately before an operation, in renal cases, I made no further ureteral examination, but the urine secreted in 24 hours was now collected and submitted to Dr. Ogden, whose report upon it follows:

"On June 10th an examination of the *twenty-four hour* urine showed the following:—Twenty-four hour quantity, 1150 cc.; color, pale, turbid; reaction, acid; sp. gr., 1013; urea, 1.13 per cent. or 12.99 grammes in 24 hours; albumin, a slight trace; bile and sugar absent. The sediment consisted chiefly of pus which was free and in clumps. Considerable squamous and scaly epithelium and a few blood globules; a few small round cells. An occasional hyaline and granular cast with renal cells and little blood adherent.

The greater part of the pus seemed to come from the same source as the squamous and scaly epithelium, in other words, probably from the vagina. I could not be certain that some of the pus did not come from the bladder or from the diseased kidney. There was still evidence of a tubular disturbance (shown by the casts) which had more the appearance of a mild renal congestion than any primary disease of the kidneys.

The sediment of this twenty-four hour urine was thoroughly examined for tubercle bacilli, but with a negative result."

June 24th I removed the right kidney by lumbar nephrectomy. The kidney was brought to the surface with great ease and rapidity and was so wholly normal in appearance that nothing but the absolute certainty which I thought myself to possess of its diseased condition would have induced me to remove it. The ureter was thoroughly normal in appearance, was cut some two inches below the kidney, closed in by catgut sutures, and dropped into the wound. On splitting the kidney after its removal the tubercular disease was at once evident, in the shape of numerous miliary tubercles on the mucous membrane of the pelvis.

The very beautiful painting which is here reproduced was made for me by Miss Florence Byrnes, artist to the Harvard Medical School.

Dr. Whitney kindly examined the kidney, and his report and the letter which he sent me are inserted below:

"The kidney was of normal size. The capsule stripped off easily, showing the surface marked by numerous small, superficial cicatricial depressions. The cortical part was slightly narrow and pale. In the papillary region were a few scattered minute opaque dots, and the pelvis was quite thickly covered with them, and markedly injected. Microscopic examination: The epithelium of the cortical tubules was low and irregular, and the cicatricial depressions were marked by a round-cell infiltration and disappearance of the tubules at that point. The opaque dots were composed of small round and epithelioid cells with an occasional giant cell. The centres were cheesy degenerated. The diagnosis is a miliary tuberculosis of the pelvis and kidney."

MARBLEHEAD, MASS., *August* 13, 1898.

Dear Dr. Reynolds:—I enclose the report of the kidney which you desired. The case is certainly favorable, as the local lesions are comparatively slight, and it is interesting that, with so little ulceration and loss of substance, a positive result should have been obtained from the inoculation with the urine.

Yours sincerely,

W. F. WHITNEY.

The patient's convalesence from the operation was rapid and satisfactory. She passed from thirty to forty ounces of urine uninterruptedly and never had a bad symptom. I have not seen her since, but a letter from Dr. Proctor, dated October 10, 1898, informs me that she is now passing forty ounces of normal urine with no evidence of renal irritation in the sediment. She has gained in flesh and color and is greatly improved in general condition.

The negative results of two examinations of the sediment for tuberculosis, made by an expert at widely separate times, and the positive result of the inoculation of a guinea-pig by one of these same sediments, with the confirmation of this positive result by inspection of the kidney in question, and the improvement of the patient's health after the operation, form certainly a picture of considerable clinical interest, more

Miliary Tuberculosis of Kidney.

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especially, as this is, so far as I am aware, the first case in which this test has been used for the examination of each kidney separately.

As so little is known of frequency of unilateral and primary renal tuberculosis I had hoped that an examination of the records of a large number of autopsies, performed upon subjects who died from other diseases than tuberculosis, might yield something of interest, and accordingly requested Dr. John T. Bottomley to examine the pathological records of the Boston City Hospital for some years past. He looked over 3300 reports which were consecutive except for the omission of deaths from tubercular disease in other organs of the body, but found only two cases of primary renal tuberculosis, both unilateral, one of which died from fracture of the spine, the other of uremia.

His abstracts of the cases are as follows:

Case I. Surg. Rec. C., Vol. 23, Page 248. Male, 48 years. No venereal diseases, case of scalp wound, alcoholism, and fractured spine.

Autopsy Rec., Vol. 16, Page 56. Autopsy showed that right kidney was about normal size; upper third replaced by several sacs, each corresponding to a pyramid and its accompanying cortex; each sac had a thin, firm capsule which was filled with opaque, white, cheesy or putty-like material, a little gritty to the touch. In one of the lower pyramids was a similar sac; no evidence of any inflammation; microscopic examination negative; pelvis and ureter normal. Bladder and appendages were normal except a few small calcified nodules in prostate.

Anatomical diagnosis. Stenosis of aortic valve. Chronic passive congestion of spleen and kidneys. Chronic tuberculosis of right kidney. Fracture of spine.

Case II. Med. Rec., Vol. 175, Page 249. Female, 45 years, married. Always well till three months before; all symptoms pointed to disease of the kidney.

Autopsy Rec., Vol. 3, Page 125. Autopsy. No truly normal tissue remained of left kidney. The entire wall was composed of abscesses of varying sizes, containing a thick almost cheesy pus. Peri-nephritic fat adherent to the wall, also to diaphragm; nothing important in other organs.

[The second case could probably have been saved by nephrectomy had the diagnosis been made a few months before. She, however, entered the hospital in the year 1880 and in a dying condition.]

LAPAROTOMY FOR INTESTINAL PERFORATION IN TYPHOID FEVER.

A REPORT OF FOUR CASES, WITH A DISCUSSION OF THE DIAGNOSTIC SIGNS OF PERFORATION.

BY HARVEY W. CUSHING, M. D., *Resident Surgeon, The Johns Hopkins Hospital.*

The present communication is based upon four recent cases of laparotomy for perforating typhoid ulcer, in one of which the abdominal cavity was opened on three successive occasions with recovery.

The fact that surgical intervention offers practically the only hope in these cases seems to be studiously overlooked, if we are to judge by the paucity of occasions in which laparotomy has been performed for this condition.

On a recent visit to the military hospital at Fort McPherson, the writer was told that of thirty autopsies held upon fatal cases of typhoid which had occurred there, perforation was found to have been the cause of death in six instances, one being of the appendicular variety. This would attribute to perforation alone 20 per cent. of the fatalities, a percentage which corresponds with that of Hare of Brisbane, and is almost twice that of Murchison (11.38 per cent.), and three times the figures given by Dr. Fitz (6.58 per cent. in 4680 cases). In none of these cases had operative intervention been advised, nor do I know of a single instance of operation for the relief of typhoid perforation on any of the possible 2000 cases which have died from typhoid in the field hospitals and elsewhere during the late war.

If the recent statistics of Gesselewitsch and Wanach (Centralblatt für die Grenzgebieten der Medizin und Chirurgie, Bd. I, No. 6, p. 382, 1898) are to be relied upon, namely, that 10 per cent. of the entire number of fatalities in typhoid are due to perforative peritonitis, we may credit 200 of these deaths to this cause alone. According to the statistics from Fort McPherson and those of Hare, 400, or double the number, would be accounted for in this way.

In a recent communication Dr. Nicholas Senn writes: "Strange as it may seem, having seen hundreds of cases of typhoid fever during the war, I was called upon only once to operate for perforation. In that instance the patient was moribund, and I refused to operate. I have reason to believe that this complication was frequently overlooked."

We have four widely different but quite characteristic histories to report:

In Case I an early diagnosis and immediate operation, before peritonitis set in, led to recovery.

In Case II general purulent peritonitis with three perforations was present, and it should be considered that the relief of the general peritonitis, and not typhoid perforation which had taken place many hours before, was the objective point of operation.

In Case III what may be considered as pre-perforative symptoms of peritonitis were present and were neglected. Perforation subsequently occurred with, unfortunately, a virulent streptococcus infection, which proved fatal despite early laparotomy.

Case IV illustrates one of the strange attacks which closely simulate perforation. No lesion was found at the operation, and the exploration had no appreciable effect upon the subsequent course of the fever.

CASE I. Surgical No. 8009. *Typhoid perforation at end of second week. Laparotomy. Suture of perforation. Drainage. Fæcal fistula after 3 days from second perforation. Spontaneous closure of fistula. Seven days later symptoms of perforation. Laparotomy. No perforation found. Obstruction over-*

looked. Two days later laparotomy for acute intestinal obstruction with closure of second perforation. Recovery.

Herbert H., aged 9, was brought into the medical wards of the Hospital on the 8th of August with the history of having been ill since the first of the month with "pain in his head and stomach." His mother and one brother were also in the hospital, and one brother had just died of "typhoid" at home. On entrance, the temperature was 104.2°, the pulse rapid, the spleen enlarged and the general appearance typhoidal. There was some tenderness noted in the lower right quadrant of the abdomen. There was no Widal reaction obtainable at this time, nor had there been in the case of his brother and mother, who had had a very mild type of fever. The patient was put on the usual bath treatment. August 9th, leucocytes 8400.

Dr. Thayer's note on August 11th says: "Abdomen is a little full. Patient does not flinch on pressure."

There is no further note of unusual interest. The boy was dull, and seemed to be having a rather severe attack. He complained much of pain in his abdomen on being given his tubs.

On the morning of August 13th (five days after admission) the patient was found to be complaining of abdominal pain. He had vomited twice and his pulse rate had increased. The leucocytes were 9600.

He was seen, in consultation with Dr. Thayer, at 1 P. M. At this time his respirations were 34; his pulse 165, rather thready and of poor quality; temperature 105°. He was crying out and complaining of general colicky abdominal pain. He was very restless; his expression pinched; his color quite cyanotic, with lips blue and extremities blue and cold. The abdomen was quite soft and there was no muscle spasm. There was considerable general tenderness, which seemed more marked on the right side. Pressure per rectum in the recto-vesical cul-de-sac seemed to cause especial pain, but no more marked on one side than the other. There was some apparent increase of dulness in the flanks, but no shifting dulness. There was no obliteration of liver dulness. The leucocytes were 16,000. The patient was immediately transferred to the operating-room.

Before the administration of anæsthesia the temperature was 105° (there had been no fall up to this time) and the pulse 170.

Operation I.—August 13th, 1898, 1.30 P. M., four hours after first symptoms, under primary chloroform anæsthesia.

Median laparotomy. Partial evisceration. Closure of perforation. Toilet of peritoneum with salt solution irrigation. Drainage.

An incision was made in the median line below the umbilicus. On opening the peritoneal cavity a considerable amount (perhaps 200 cc.) of sero-purulent fluid escaped, coming chiefly from the pelvis. Cultures were taken from this fluid and it was also immediately examined in cover-slip preparations. It contained a great number of polymorphonuclear leucocytes, but no micro-organisms could be found. The serosa of the intestine was everywhere greatly congested, but evidently the greatest reaction was in the right iliac fossa, where the loops of the ileum were especially injected and covered with a slight fibrinous deposit. The general cavity to the left was walled off with gauze and the loops of the ileum drawn from the wound. The last foot of the small intestine showed several greatly thinned areas corresponding to Peyer's patches. In the centre of one of these areas, the surroundings of which were quite bluish in color, was a small perforation about two millimetres in diameter, from which fluid fæces were flowing. It was about 25 cm. from the cæcum and situated in the free surface of the bowel. A few centimetres beyond this were two more patches, which seemed very thin and practically covered by little more than serosa. A few fine, filmy adhesions held the omentum to this part of the bowel.

The perforation was closed by a circular suture of fine silk taken about the edge of the thinned area, which was fortunately small, measuring about one centimetre in diameter. A similar inversion of the two thin neighboring patches would have been attempted, but the patient's condition at this time demanded immediate attention and the idea was abandoned. His pulse was almost imperceptible and his respirations were very shallow. Hypodermic injections of strychnia were given and an infusion of a litre of salt solution in the pectoral region. The exposed coils of intestine were irrigated and the abdominal cavity was flushed out with salt solution. A strip of bismuth gauze was left in for drainage, leading down to the site of the suture, and another to the two thinned Peyer's patches. The omentum was pulled down over the gauze. The abdominal wound was then partly closed. The operation lasted but twenty minutes from the beginning of anæsthesia till the final closure.

The cultures taken from the fluid found free in the peritoneal cavity remained sterile. No cultures were taken from the material flowing from the perforation.

The patient rallied well from the operation, and in eight hours the pulse had fallen to 112, and the temperature to normal. There was no vomiting, and he slept most of the time for the next twenty-four hours, taking liquid nourishment (albumen water) well, during his waking intervals. Three hundred cubic centimetres of salt solution were given *per rectum* every four hours for thirst. On the following day the temperature again became elevated; the day after the leucocytosis disappeared, and for the following ten days the clinical picture was that of an ordinary typhoid in the third week. A fæcal fistula developed on the second day, discharging typical pea-soup fæces. It was impossible to tell, at that time, whether this discharge came from the broken down suture or from one of the neighboring thinned Peyer's patches, to which the drain led. (It was subsequently proved to come from the latter.)

The boy complained a good deal of abdominal pain at times, and of pain on micturition, but there was no abdominal rigidity or other symptoms of peritoneal inflammation. He was given regular ice sponges, after which he would sleep for long intervals. The fæcal fistula ceased to discharge after a few days. There was considerable diarrhœa at this time (cf. Clinical chart).

August 18th. (*6 days after operation I.*) A positive Widal reaction was obtained by Dr. Schenck for the first time (in ten minutes in a dilution of 1 to 40). Cultures from the rectum, taken through a high rectal tube, showed only the bacillus coli communis. The patient had a pinched look and was very peevish. The pulse was weak and dicrotic. A crop of rose spots was present on the abdomen.

August 25th. (*13 days after operation I.*) On the evening of this date, after a very good day, the patient became suddenly much worse and vomited several times. At 9 P. M., when seen in consultation with Dr. Thayer, he was quite

collapsed and very restless. His abdomen was slightly distended. He was hiccoughing and complaining of abdominal pain. He looked pinched and the extremities were cold and sweating. His leucocytes were only 4000.

A definite diagnosis of perforation was made and steps taken for immediate operation. An hour later the temperature had fallen four degrees to 98.6°, and the leucocytes had increased to 13,000.

Operation II, August 25, 1898, 10.30 P. M.; two hours after first symptoms; under chloroform anæsthesia. *Median laparotomy. No cause for symptoms demonstrable. Closure with drainage.*

The abdomen was opened beside the first incision, avoiding the site of the fistula. There was no free fluid. The bowels were not injected except to a slight degree in a few places, corresponding to some of Peyer's patches. No evidence whatever of a perforation could be found. There were no adhesions except about the loop of ileum which led to the old sinus and which was surrounded by quite firm omental adhesions. These were not broken up. There was no particular distension of one coil more than another. (There was nothing to suggest the thought of obstruction, and I must confess it did not occur to me. I supposed that I had overlooked a perforation somewhere, but the patient's condition precluded further search.) The wound was closed, with a small drain leading to the omental adhesions.

The operation lasted thirty-five minutes from the beginning of anæsthetization. Cultures taken from the site of the adhesions remained sterile.

The patient's condition did not seem to be materially affected by the operation. The collapse, with cold sweating extremities, the sudden onset of abdominal symptoms, with hiccough, vomiting and pain, the drop of temperature without signs of hæmorrhage and the rapid rise in the leucocytes made operative intervention imperative.

During the following twenty-four hours the condition became progressively worse. All attempts at feeding were followed by immediate vomiting, and enemata were but partially retained. The patient had voided no urine for 18 hours. The lips were parched; the tongue dry and coated. By the early morning of August 27th (30 hours after operation II) the distension had become more pronounced, and on close inspection, with a candle placed beside the exposed abdomen, a slight visible peristalsis was to be made out, which first demonstrated that obstruction, and not peritonitis, (despite the leucocytosis at this time of 20,000) was responsible for the symptoms, and it was learned that the irrigations, preceding the enemata, since the time of collapse, had not been stained with fæcal matter as had previously been the rule.

The child's condition, however, seemed to forbid operative intervention. He was vomiting without effort, restless and in collapse more pronounced than at any time previous. The radial pulse could not be counted. He was given small doses of morphia and strychnia and hot compresses over his abdomen, which quieted him considerably, and four hours later an operation was determined upon.

Operation III, August 27th, 8.30 A. M. (34 hours after operation II), under chloroform anæsthesia.

Median laparotomy. Acute intestinal obstruction due to adhesions about a second perforation. Obstruction relieved. Perforation sutured. Closure with drainage.

The recent wound was re-opened. There was no sign whatever of peritonitis. The small intestine, which was greatly distended, was turned out of the abdomen together with that loop of ileum and its surrounding omentum, which had become adherent to the anterior parietes, as a consequence of the drainage at the first operation. In this omental mass was an acute kink of the bowel, about ten centimetres proximal from the old suture, the distal part of the bowel and the colon beyond being completely collapsed. The original suture was intact. At the exact situation of the kink, and covered by the omentum which caused the obstruction, was a second perforation, apparently corresponding to the thin Peyer's patch seen at the first operation.

On freeing the obstructing omentum, gas and fæces in great amount escaped from this perforation, greatly diminishing the distension. The perforation was about 1 cm. in diameter, with rounded edges, showing everted mucous membrane, so that evidently the whole floor of the ulcer had given way. It was closed easily by a single purse-string suture of fine silk. The bowels after irrigation with salt solution were replaced. A small drain of bismuth gauze was inserted down to the wad of omentum, from which cultures had previously been taken. (In these cultures Dr. Clopton found an abundant growth of the bacillus coli communis, of proteus vulgaris, bacillus lactis aërogenes and an organism culturally closely akin to the bacillus typhosus.) The abdominal wound was partially closed.

The operation lasted twenty-five minutes from the beginning of anæsthetization. The patient's pulse could not be counted. He was given 500 cc. of salt solution in each pectoral region and also *per rectum*.

Throughout the day the child's condition remained most serious. Distension was pronounced with active, visible and painful peristaltic cramps. He was given small doses of morphia, and large hot bichloride fomentations, for which he seemed very grateful, were at short intervals placed over the whole abdomen. He vomited only twice after returning to the ward, and soon began to retain his nourishment. Twelve hours after the operation, flatus first began to pass from the lower bowel. On the following day, though considerable meteorism remained, the bowels moved three times, and considerable diarrhœa persisted for the four or five succeeding days. As the clinical chart shows, the patient, though greatly emaciated and very feeble, returned to his typical typhoidal condition which ran its course in the next ten days.

Convalescence was very tedious. The child had a series of superficial indolent staphylococcus albus abscesses over the back and shoulders which had to be opened, and a protracted bloody mucous diarrhœa set in which kept him thin and weak. The abdominal incision broke down after the third operation, and was slow in healing. He has, however, made a complete recovery.

In Case I, therefore, we have what is most unusual, a child with early perforation. The symptoms at onset were typical, and the condition was readily diagnosed. The early vomiting was a great help, while the prostration, abdominal pain, tenderness and the leucocytosis made the picture complete before the onset of peritonitis.

The history of preceding abdominal pain for some days before the actual occurrence of perforation is interesting and, in the light of the subsequent cases, important, in that it most probably was associated with a slight local peritonitis due to the near approach of an ulcer to the general peritoneal surface. From what will be said later it seems possible that clinical symptoms arising from this condition are not uncommon and that they may be utilized in foretelling a perforation, or at all events in putting the attendant on his guard so that the patient may be kept more than ordinarily quiet and tubs omitted, especially if they are disagreeable and resisted.

Among other points of unusual interest is the fact that so prompt intervention was rendered possible, owing to the discernment of Dr. MacCallum, who had charge of the ward, and that the perforation was closed before there was any evidence of peritoneal infection, the cultures from the free fluid remaining sterile.

The subsequent perforation of a neighboring ulcer showed that it would have been desirable at the first operation to have turned in by a suture those patches which seemed thin, and threatened perforation, as was done by Sifton* in his case with recovery, and also by W. Hill.† In view of the fact, however, that there were several of these areas, and that it did not seem justifiable to further prolong the operation, a strip of gauze was placed leading to the two worst looking patches. Whether the trauma of the gauze was itself responsible for the subsequent perforation, or, whether by forming adhesions, it saved the general cavity from the escape of intestinal contents through a perforation, which would have occurred in any case, must remain undetermined.

The leucocytosis which at the first operation afforded an apparently certain indication was completely misleading at the second. Here, although in two separate counts the leucocytes were 20,000 there was found no inflammatory reaction to account for the increase. I do not know whether obstruction is usually associated with leucocytosis or not.‡

It is also noteworthy that the perforation was not associated with an early drop in temperature, contrary to the supposed rule, whereas one of the first symptoms of the obstruction was a fall of four degrees. The importance of immediate intervention was evidenced at the first operation. Practically the same symptoms appeared twelve days later without perforation. As was found subsequently these symptoms were due to an acute obstruction, and at the second operation, which was performed immediately after the onset of symptoms, the real condition was overlooked because at this early period distension of the proximal bowel which would naturally have suggested obstruction had not yet taken place. The question of justifiability of such an immediate intervention on the first symptoms of perforation will be considered later, as well as the difficulty of distinguishing between acute obstruction and perforation in their early stages.

It was learned from this case that a median incision was a bad one as the lesions occur in the right iliac fossa almost as naturally as do appendicular ones. It is also apparent that the mere performance of laparotomy in the course of typhoid fever, provided it is made before the occurrence of septic extravasation, is in itself attended with little more risk than a similar procedure in febrile states, the only apparent drawback to it being the necessary omission of any bath treatment during the subsequent progress of the fever.

I have found in the literature only one instance in which more than one laparotomy has been performed in an attempt to combat intestinal perforation and its sequelæ. In Bogart's* interesting case a perforation in the ileum, which was closed by the tip of the appendix, occurred in a third attack of fever. Death resulted three days later from obstruction at the hepatic flexure of the colon, an operation for the relief of which was abandoned. Of this case Keen† says: "I can scarcely think that we would ever be justified in re-opening the abdomen in such a case. Possibly a very exceptional case might justify such a procedure, but a typhoid patient rarely escapes with his life even after one operation and could not be expected to survive a second. The same remark would apply to any new perforation which might occur. Such cases must unfortunately be left to their fate; but if the surgeon has been careful to search for and suture any impending perforation he has done much to prevent such a disaster."

Dr. Keen's last remark holds true, but it is hard to agree with the statement that a patient should be left to his fate, no matter how desperate the condition, provided surgical intervention offers any chance of relief, forlorn though it may be. The great vitality of some of these patients is illustrated as well by this case as by the remarkable one of Dr. Finney's in which after two relapses, an otitis media, a pleurisy, and phlebitis subsequent to the operation, there was eventually a complete recovery.

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*Sifton, H. A.: Chicago Clinical Review, Vol. IV, p. 368, 1894–5.

†Reported by Keen: Surgical Complications and Sequels of Typhoid Fever, 1898, p. 238, Case 40.

‡In a condition of obstruction at the hepatic flexure, following a recent operation for acute appendicitis the patient had a leucocytosis of 44,000 without peritonitis, whereas during the acute stage of his appendicitis the leucocytes had been only 23,000. A differential diagnosis between peritonitis and obstruction is most difficult. Bogart's case was similar to this one, an obstruction simulating general peritonitis. Damner Harrison (Brit. Med. Jour., Oct. 20, 1894, p. 865) operated on a typhoid case for perforation, and found an obstruction. Barbe (Étude clinique sur certaines formes des perforations de l'intestin grêle: Importance du diagnostic précoce. Thèse de Paris, 1895) calls attention to similar cases. No record of the number of leucocytes is given by these writers. The high degree of leucocytosis, such as was present in the writer's two cases, may possibly be of some diagnostic value in differentiating between these conditions, though in a manner entirely opposed to the usual interpretation.

*Bogart, J. Bion: Laparotomy for Perforating Typhoid Ulcer of the Ileum, etc. Annals of Surgery, Vol. 1, 1896, p. 596.

†Keen, W. W.: Surgical Complications and Sequelae of Typhoid Fever, p. 232, 1898.

CASE II. General No. 23,970.—*Typhoid perforation in the fifth week. Laparotomy under cocaine anæsthesia. General peritonitis. Suture of three perforations in ileum. Death four hours after operation.*

September 3, 1898. William N., aged 18, was brought to the medical wards of the hospital with the history of a febrile attack of three weeks' duration. The patient's mother and sister died at home during his stay in the hospital of "malignant typhoid."

The patient was dull and stupid, and presented a typical typhoidal appearance. The medical note on the abdomen at entrance is as follows: "*Abdomen.* Peculiarly mottled, especially in inguinal regions, a bluish discoloration, taches bleuâtres. Walls somewhat tense; grooves obliterated; everywhere tympanitic. Slight tenderness across upper abdomen in umbilical and epigastric regions. Spleen enlarged, edge readily palpable. *Liver.* Relative dulness at upper border of 6th rib; absolute in 6th space and extending to costal margin; towards left extending 3 cm. below xyphoid; edge palpable."

Sept. 5th. The abdomen was noted by Dr. McCrae as being natural.

Sept. 10th. The patient has well-marked diarrhœa with colicky pains.

Sept. 11th. Widal reaction positive in dilution of 1 to 100.

Sept. 12th. Dr. McCrae. "Patient has been complaining of abdominal pain. Abdomen is slightly distended, tense. Rose spots present. Liver dulness present. No abdominal tenderness on palpation. Leucocytes 7500 at 10 A. M." 4 P. M. Patient vomited twice in early afternoon after nourishment. Leucocytes 8400 at 8 P. M.

Sept. 13th. "Patient has been very ill for 48 hours. Eyes are sunken. He has somewhat the look of collapse. Abdomen is somewhat full with respiratory movements present. The walls are very tense and tender on palpation. Liver dulness absent in mammary line, present in mid-axillary line."

The patient vomited after nourishment at 10 and 12 o'clock last night.

The leucocytes on this date were as follows:

Leucocytes at	9	A. M.	6000	
"	" 10	"	7200	
"	" 11	"	8800	
"	" 12.15	P. M.	6400	
"	" 1.30	"	6000	operation performed.
"	" 2 00	"	7000	

At 1 P. M. a note by Dr. Thayer is as follows: "Patient lying on back. Tongue beefy, eyes sunken and wide open. Respirations short and shallow. Patient has a peritonitic facies. Green vomitus in sputum cup. Pulse small, 152. Abdomen tense, full and tympanitic. With patient on his back there is no hepatic flatness in the mammary line and none till one almost reaches the axillary line. There is dulness in either flank which disappears when the patient lies on the opposite side. Frequent vomiting during the examination. There is a well-marked friction throughout the right axilla which is heard all the way down to the costal margin. With patient lying on his left side there is no hepatic flatness anywhere.

The patient was immediately taken to the operating-room.

Operation at 1.30 P. M. under cocaine anæsthesia. *Closure of three perforations in ileum. Irrigation and drainage for general septic peritonitis.*

A linear infiltration of the skin with Schleich's solution was made in the right linea semilunaris. On opening the abdominal cavity there was an explosion of gas followed by the escape of a large amount of stinking material looking like pea-soup stools. The bowels were of a dark bluish color, distended, covered by a thick plastic lymph and everywhere bathed in the fœcal extravasation.

The cæcum was located, and the first loop of ileum when drawn out showed three large ragged holes about 1½ cm. in diameter and with fine bridges across them made by threads of submucosa.* They were closed with Halsted mattress sutures. The appendix and colon were free from perforations. The patient was given a few whiffs of chloroform; the bowels were turned out, and the abdominal cavity cleaned as thoroughly as possible and irrigated with salt solution. Drainage was left in to the bottom of the pelvis and to the site of the suture.

The operation lasted 20 minutes.

Needless to say his condition was desperate with a pulse of 160 and respiration 60.

The patient rallied somewhat after the operation under stimulants and salt infusion, but remained in a state of euthanasia, often seen in severe septic infections, and died four hours later. Unfortunately no post-mortem examination could be made.

The cover-slip preparations taken from the general cavity at operation showed a great diversity of organisms, some cocci, but mostly bacilli of various shapes and sizes, and a great number of pus cells. No streptococci were seen. Nothing was grown out on culture but the bacillus coli communis.

This case well illustrates the practical hopelessness of operation when perforation, at its onset, has been overlooked, and the operation delayed until the stereotyped symptoms of extensive extravasation, such as obliteration of liver dulness and evidences of shifting free fluid, have appeared. It is such cases as this which render the operative statistics for perforation so uniformly bad. Abbe's† case, however, makes recovery seem never impossible after operation.

The complete absence of abdominal tenderness and of leucocytosis unfortunately was misleading, but the preceding diarrhœa, abdominal pain and vomiting under ordinary circumstances would have led to early exploration had it not been for the fact that so many of the house cases this fall have complained of adominal pain and tenderness associated with diarrhœa. This point will be referred to later.

The importance of making cover-slip preparations as well as immediate plate cultures from the abdominal contents in cases of peritonitis is well shown by the fact that the bacillus coli communis overgrew all other organisms in what the cover-slips had shown to be a polyinfection. Careful investigations, such as those of Flexner‡, show what a variety of organisms may be present. Undoubtedly the colon bacillus being more in evidence is frequently held responsible for peritoneal infection due to more virulent but culturally less vigorous organisms. In one of Flexner's cases (Case IV) there was obtained from the peritoneal cavity the bacillus typhi abdominalis, bacillus coli communis, proteus vulgaris,

* Such as are shown in Keen, op. cit., Plate V, Fig. I.

† Abbe, Robert: Perforating Typhoid Ulcer. Peritonitis. Operation. Recovery. Medical Record, Vol. XLVII, p. 1, January 5, 1895.

‡ Flexner, Simon: Certain Forms of Infection in Typhoid Fever. Johns Hopkins Hopital Reports, Vol. V, 1895.

staphylococcus aureus and the streptococcus pyogenes. I know of no other case in which the bacillus typhosus has been obtained in culture from a general peritonitis following perforation. The organism in our Case I, though akin to it, was not positively identified. Körte* isolated the bacillus from a general peritonitis which originated however from a ruptured suppurating mesenteric gland. Klein also is said to have obtained it, but from a localized peritonitis.

CASE III. Surgical No. 8131.—*Typhoid perforation at end of fourth week after prolonged abdominal symptoms. General streptococcus peritonitis. Laparotomy. Suture of perforation. Death after eight hours.*

Sept. 5, 1898, Peter B., colored, aged 31, was admitted to the medical wards complaining of having had "pain in his head and stomach and general weakness" since August 23rd. He had had some diarrhœa and abdominal pain during this time, but had not taken to his bed.

The note on the abdomen at entrance is as follows: "*Abdomen* looks natural; no distension; costal and iliac grooves are well marked; respiratory movements are present. Some tenderness on palpation, especially in right inguinal and iliac region. Liver dulness begins at the sixth rib and extends to costal margin. Spleen is just palpable." *Blood count.* Red corpuscles 4,820,000. White corpuscles 5,600.

The Widal reaction was positive in dilution of 1 to 100. The patient was very ill and delirious at times, and on the night after admission jumped ten feet out of the ward window without, however, injuring himself. A slight leucocytosis was found a few days after admission which, coupled with his abdominal pain, occasioned suspicion, and he was watched very closely.

On several occasions the writer saw him in consultation with Dr. McCrae, but the complete absence of objective abdominal symptoms and the fact that there were several patients in the wards with similar subjective symptoms made us hesitate about operative intervention. For ten days he pursued a usual typhoid course, though the leucocytosis at one time reaching 15,200 persisted (cf. Clinical chart) and abdominal pain was constantly complained of. There was no diarrhœa. He was given the usual bath treatment.

Dr. McCrae's note the morning of the 17th inst. states: "General condition good. There is no delirium. Tongue is still coated. Patient frequently lies with knees drawn up. He states that he has very slight abdominal pain this morning. Abdomen is flat, soft on gentle palpation. Patient complains of severe pain when pressure is applied. Muscles at times are rigid, at others soft.

I am inclined to believe that his perforation took place the following night coincident with the drop in temperature (cf. Clinical chart). His pulse at midnight was recorded at 120 and his respirations at 36. By a strange misfortune during rounds the next day at noon he seemed very much better. His pulse was 76 and respirations 22, possibly as a result of the recent tub, and there seemed to be no change in his abdominal condition.

He was quite delirious during the day. There was no nausea or vomiting His leucocytosis had disappeared. At midnight of this day Dr. McCrae found him in considerable pain, lying on his back with his knees drawn up. He was dull and answered questions slowly and unintelligently. Respirations 44. Pulse 120. There was some vomitus on the floor beside the patient's bed. This was the first vomiting that had been noted.

Respiratory movements were absent from the abdomen. There was no fulness but general tenderness on palpation, especially in the right iliac fossa. There was distinct dulness, which was movable, in the right flank. The liver dulness began at the 7th interspace and extended to the costal margin. His leucocytes were 4300.

September 19th. *Operation* at 1 A. M. *Ether anæsthesia. Laparotomy. General peritonitis. Suture of perforation in ileum. Irrigation. Gauze drainage.*

The incision was made through the right rectus sheath, and the muscle drawn toward the median line. On opening the abdominal cavity bubbles of gas and sero-purulent fluid escaped. Cover-slips from this fluid were immediately examined and found to be full of streptococci with an abundance of other pleomorphic organisms.

The ileum was quite distended. For about two feet from the cæcum, as it was withdrawn, it appeared injected and pretty extensively covered with a delicate, fibrino-plastic lymph which could be readily peeled off in sheets. This pellicle was in many places glistening and quite transparent. There were no adhesions. About 10 cm. above the cæcum in the centre of a dark bluish area measuring about 1½ cm. in diameter was a small perforation which was partially occluded by a pouting nubbin of red mucous membrane and from which there seemed to be escaping very little of the intestinal contents at this time. The perforation and thin patch were turned in with Halsted sutures. There was not a great amount of extravasation or free fluid present, certainly not enough to have given shifting dulness.

The eventrated bowel was irrigated and the fibrin wiped off with wet salt sponges. The general cavity was wiped dry with salt sponges. Drainage was inserted to the bottom of the pelvis and to the site of suture and a neighboring thin Peyer's patch.

The patient stood the operation, which lasted 30 minutes, fairly well. There was no vomiting during or after the anæsthesia.

Death supervened 8 hours after the operation. The temperature remained high. There was no vomiting and nourishment was taken frequently. Nutritive and stimulant enemata were given and retained. The clinical picture was one of acute general toxæmia, such as streptococcus infection sometimes produces.

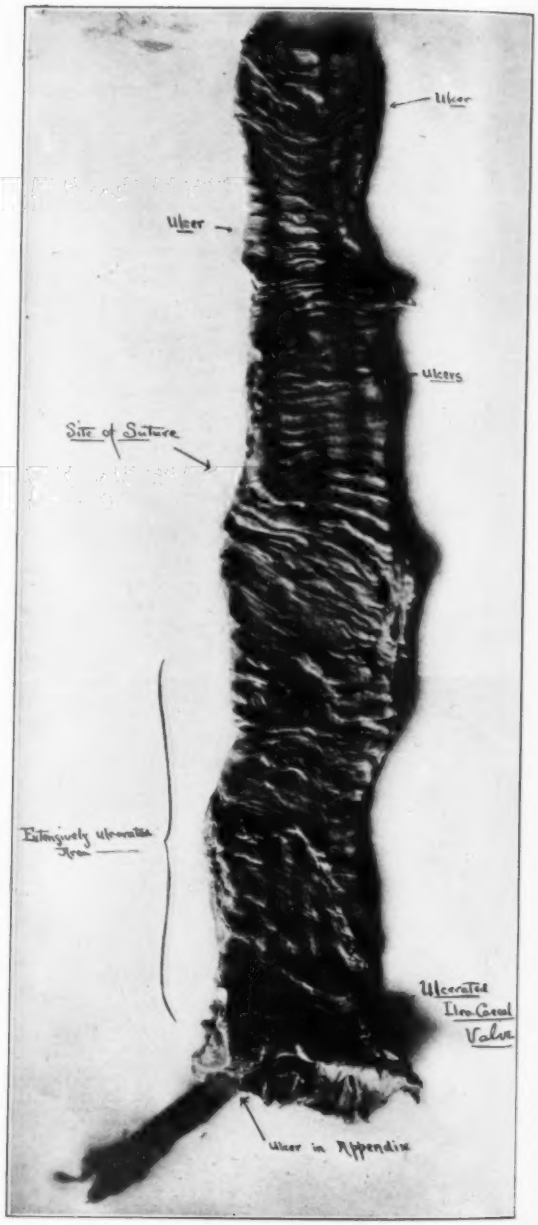
From the peritoneal fluid at operation Dr. Clopton obtained in cultures an abundant growth of the streptococcus pyogenes, the bacillus coli communis, the bac. lactis aërogenes and a yeast fungus.

Autopsy. Eleven hours after death. Dr. Nichols.

Anatomical diagnosis. Typhoid fever. Perforation. General fibrino-purulent peritonitis. Operation. Healing ulcers in ileum, cæcum and appendix. Slight ileo-colitis. Suppurating peritoneal gland. Acute splenic tumor. Cloudy swelling of kidneys, etc., etc.

The protocol need not be given here in full. The lesions in the organs were typical of typhoid infection. The peritoneal surfaces were quite generally involved in an inflammatory process with thin adhesions and without the production of much fibrin or pus. The free fluid had a sero-purulent character. There was a second threatening perforation a short

* Körte, W.: Erfahrungen über die chirurgische Behandlung der allgemeinen, eitrigen Bauchfellentzündung. Verhandlungen der deutsch. Gesellschaft für Chirurgie, 21ter Congress, p. 164, 1892.



PHOTOGRAPH OF THE ILEUM OF CASE III.

distance above the ileo-cæcal valve. The mesenteric glands were all swollen, soft, and one showed an area of suppuration the size of a pea. There were two or three small round healing ulcers in the cæcum and appendix. The following description is taken directly from the protocol:

The Ileum presents a remarkable appearance. The very edge of the ileo-cæcal valve has preserved its mucous membrane. Above this the mucous membrane has been completely destroyed over a large surface by single, confluent, often suspicious looking ulcers. One has reached the size of 7 cm. in length by 4 cm. in breadth. (This is well shown in the photograph.) The central part of this has a small clot adherent to it, and corresponds to a hæmorrhagic area on the serosa, looking like an imminent perforation situated 4 cm. above the ileo-cæcal valve. These ulcers have the same general characteristics, their edges are raised, seem opaque, congested and partly hæmorrhagic. Their base is clean and apparently extends down to the transverse musculature. It is of bright red color, and transverse striae can be plainly seen. About the edges and often running in small strands across the base is a pink, delicate, new growth of epithelium. The general direction of these ulcers is longitudinal. This extensively ulcerated area does not extend much further than 11 cm. from the valve. Fourteen centimetres above the valve is a similar ulcer, puckered and inverted by the silk sutures where the perforation had occurred. This ulcer was not larger than a five-cent piece.

There are about five small, similar ulcers at various distances apart above this. The whole mucous surface is somewhat congested and there are small sub-mucous ecchymoses. Peyer's patches and the solitary follicles are not swollen to any appreciable extent.

Bacteriological Report—Dr. Harris.—Cultures from the abdominal cavity gave the streptococcus pyogenes. From the spleen and gall-bladder was obtained an actively motile bacillus, which decolorized by Gram, and in cultural characteristics corresponded to the bacillus typhosus. From the liver, kidney and peritoneal gland an organism was obtained which was identified as the bacillus coli communis. Also from the pelvic exudate, lung and peritoneal gland was obtained a bacillus corresponding to the bacillus lactis ærogenes.

This case would appear to be of special importance in that it exemplifies the existence of a definite recognizable condition spoken of above as the pre-perforative stage of ulceration. It seems not unnatural to suppose that, owing to the extreme degree of ulceration of the ileum or possibly of the appendix, some inflammation of the serosa, limited by adhesions, may have taken place. This would account for the abdominal pain, tenderness and leucocytosis of several days duration and the disappearance of the latter after perforation had actually occurred. Doubtless it would have been better to have operated early, and have sutured or drained from any suspicious patch.

These preliminary abdominal symptoms undoubtedly somewhat disguised those of perforation with extravasation when it subsequently actually occurred, and the abdomen unfortunately was not opened until after evidences of general peritonitis had begun to appear.

How often a streptococcus peritonitis has been the cause of death in the fatal operative cases cannot be told, and it is a matter of regret that bacteriological reports showing the variety of peritonitis present in these cases are not more often noted. Keen* says: "There is but a single instance, so far as I know, of a bacteriological examination of the contents of the peritoneum in typhoid perforation." Undoubtedly peritonitides of this nature are very fatal, and the abundance of long streptococcus chains, found in cover-slip preparations during the operation, gave immediately a bad prognosis to what seemed otherwise a favorable case.

The extent of peritonitis macroscopically was one such as is not infrequently recovered from in those more fortunate cases in which streptococci are not the paramount infective agent. As Durham† has emphasized, "the more virulent the infection the less marked are the local signs of peritonitis."

A streptococcus infection in typhoid is undoubtedly a very severe complication and Vincent‡ believes that it carries with it an extremely grave prognosis. Doubtless, considering its frequency in autopsy records of perforation, it has been present in many of the fatal cases which have succumbed after operation. Reports by Flexner,§ Fraenkel‖ and others show how frequently streptococci are obtained from the peritoneal exudate at post-mortem examinations after typhoid perforations. Tavel and Lanz¶ in their extensive report on peritonitis, recognize the frequency and importance of streptococcus infections, but they seem to have encountered no cases of typhoid perforation at the surgical clinic in Bern, nor has Körte** in his recent paper added any to his two previously published cases.

It is strange with the degree of ulceration found and the abundance of streptococci present, that there was no diarrhoea in this case. The steady drop in the leucocytes, after the perforation and with the onset of general peritonitis, is a most interesting feature and recalls the condition in Case II, where with the purulent peritonitis no leucocytosis was present, though it will be observed that no count was made in the latter case at the time of probable perforation or just before it. It is quite well recognized that in appendicitis the leucocytosis, which may be high (20,000 to 30,000) before, drops after perforation, and

* Op. cit., p. 220.

† Durham: On the Clinical Bearing of Some Experiments on Peritoneal Infections. Med. Chir. Trans., London, Vol. LXXX, p. 191, 1897.

‡ Vincent, M. H.: Étude sur les résultats de l' association du streptocoque et du bacille typhique. Annales de l' Institute Pasteur, Vol. VII, p. 141, 1893.

§ Flexner, Simon: Certain Forms of Infection in Typhoid Fever. Johns Hopkins Hospital Reports, Vol. V, 1895.

‖ Fraenkel, Eug.: Zur Aetiologie der Peritonitis. Münchener med. Wochenschrift, Bd. XXXVII, s. 23, 1890.

¶ Tavel, E. und Otto Lanz: Ueber die Aetiologie der Peritonitis. Mitteilungen aus Kliniken und medicinischen Instituten der Schweiz, 1898.

** Körte, W.: Weiterer Bericht über die chirurgische Behandlung der diffusen eiterigen Bauchfellentzündung. Mitteilungen aus den Grenzgebieten der Medizin und der Chirurgie, Bd. II, 1897, p. 145.

with the onset of general peritonitis often disappears completely.

I am inclined to believe that in these suspicious cases the tubs should be discontinued. The late appearance of vomiting and its single occurrence shows the unreliability of this symptom for the diagnosis of perforation.

CASE IV. Surgical No. 8154.—*Typhoid fever in fourth week during relapse. Supposed perforation. Exploratory laparotomy negative. Recovery.*

Maggie P., aged 15, was admitted to the medical wards, August 8, 1898, in the first week of typhoid fever. The fever pursued a typical course (cf. Clinical chart) without abdominal symptoms, with no leucocytosis and with a positive Widal reaction. A moderately severe phlebitis of the left leg appeared unassociated with leucocytosis on the 33rd day. There was considerable swelling of the leg, and tenderness over the femoral vessels. The temperature reached 104°, but in a few days dropped to normal, and remained down twelve days. The patient was up in a wheel-chair, and without symptoms.

On September 22nd (the 52nd day after onset) a relapse of the fever came on abruptly, the temperature rising suddenly to 103° and on the following day to 105°. There was some nausea and vomiting, without abdominal tenderness or pain, but associated with a leucocytosis of 16,000. This condition persisted for the succeeding 48 hours, and the right iliac fossa was noted as being slightly resistant. Some tenderness was noticed in the right calf.

On September 24th, the child began to complain of abdominal pain. She was seen in the evening in consultation with Dr. McCrae, but there were no objective abdominal symptoms at that time. The condition was much as on the preceding day. There was some tenderness in both calves on pressure.

September 25th, 1.30 A. M., the child awoke crying out with a sudden sharp pain in the abdomen "unlike anything she had previously had." Some nausea and slight vomiting followed. Leucocytosis 11,000. Two hours later, 3.30 A. M., the patient was again seen by the writer. She was complaining of colicky pain in the abdomen. Her thighs were kept flexed. There was some slight distension present. The liver dulness did not reach the costal margin by a finger's breadth. The walls were somewhat tense, and with moderately deep pressure in the right iliac region muscle spasm was elicited and the patient would cry out with pain. The chief rigidity, however, was above the level of the umbilicus. Her leucocytes had dropped to 8200. She vomited about 20 cc. of nourishment given her just before.

Operation September 25th, 6 A. M., four and one-half hours after the first appearance of symptoms.

Exploratory laparotomy. Negative findings. Closure without drainage.

An incision was made in the right iliac region. There was no free fluid in the general cavity; no injection of the peritoneum. The appendix, ileum and ascending colon appeared normal and without adhesions. The Peyer's patches in the ileum were swollen and hard, being felt like buttons through the bowel. The gall-bladder was not distended or inflamed. The pelvic viscera were negative. No thrombi could be palpated in the internal iliac veins. There were no suppurating glands felt in the mesentery. The abdominal wound was closed. The operation lasted only fifteen minutes.

The patient showed no ill effects from the operation. There was no subsequent nausea or vomiting; her leucocytosis disappeared, and she passed through an uneventful relapse of fever (cf. Clinical chart).

In the light of our previous experience with Cases II and III and with such symptoms as sudden acute abdominal pain, nausea and vomiting with increasing distension, some rigidity and tenderness and leucocytosis (especially a falling leucocytosis), the writer did not dare take the responsibility of withholding operative intervention even though there was some doubt as to the diagnosis. The possibility of an extension of the phlebitis to the internal iliac veins, thus causing some abdominal pain and tenderness, was thought of, but with the preceding phlebitis of September 22nd there had been no leucocytosis, pain or vomiting. The fact that the chief rigidity was above the level of the umbilicus suggested a gall-bladder complication, which in itself would have demanded exploration. The general appearance of the child was not that of collapse following perforation. There was no marked change in pulse, temperature or respiration with the above-mentioned symptoms. Nevertheless, I believed much less responsibility to be associated with an exploratory laparotomy than with running the risk of neglecting a perforation until signs of peritonitis should occur.

The precise cause of the patient's symptoms remains undecided. She was a very nervous child, and there had been some children with acute abdominal affections in the ward during her previous period of convalescence.

Similar cases have been reported. Herrington and Bowlby* report an operation on a young girl convalescing from typhoid who had even more marked symptoms of perforation with collapse than those related above. There is no mention of a leucocyte count. A laparotomy revealed no peritoneal lesion. Convalescence was uninterrupted.

General Considerations on Operation for Typhoid Perforation.—In recent years several tables have appeared in which are included all of the supposed authentic cases of operation for this particular complication in typhoid, notably those of Finney, Keen and Monad and Vanverts.

Statistics, however, always misleading, are especially so when they concern a question involving so many considerations as are included under the one head of "Results of Operation for Typhoid Perforation."

In the first place two distinct varieties of perforation may be recognized in which the operative prognosis is widely different. In one, the appendicular form, the process takes place in a quiet corner of the abdomen usually remaining localized, owing to the formation of adhesions, for perhaps a long time. In these cases some pre-existing chronic appendicular trouble may predispose toward perforation in the same way as does an ulcer of typhoidal origin in this situation. The condition, then, is practically one of acute perforative appendicitis occurring in the course of typhoid, and has the same prognosis and surgical features as similar conditions unassociated with

*Herrington, W. C., and Bowlby, A. A.: Typhoid Fever Convalescence; Symptoms of Perforation, Laparotomy; no Lesion found; Recovery. Med. Chir. Transactions, London, Vol. LXXX, 1897, p. 127.

typhoid which give a certain percentage of recovery in unoperated cases.

In the other variety the perforation almost always occurs in the freely moving bowel, usually in the lower foot of the ileum.

I believe that Dr. Fitz* first clearly distinguished between these two varieties of perforation in typhoid, emphasizing the fact that many cases would be called appendicitis which, when occurring during typhoid fever, are classed as perforations. Undoubtedly the appendicular cases are much more common than has been ordinarily supposed. Fitz finds, however, only 3 per cent. in 167 cases. Of the 20 cases of perforation in the pathological records of the Johns Hopkins Hospital there have been 2 appendicular perforations which, grouped with a single case out of the nine of which I am personally cognizant, makes 9.6 per cent. which have occurred in the appendix.

To further quote from Dr. Fitz's paper: "The probability of its occurrence (perforative appendicitis) furnishes the best solution to the prognosis of intestinal perforation in the latter disease (typhoid fever). Most cases of recovery from symptoms of perforation of the bowel in typhoid fever are those in which an attack of appendicitis is most closely simulated, while the fatal cases of perforation of the bowel in typhoid fever are, in the great majority of instances, those in which other parts of the bowel than the appendix are the seat of of perforation."

It is of course important to recognize the fact that either of these conditions may be present, but a differential diagnosis can hardly be made, and were such possible, operative interference is as surely indicated as in any acute appendicitis. The prognosis in the appendicular varieties, for the reasons given above, is naturally more favorable, but in all cases the earlier surgical intervention is sought the better for the patient. This applies especially to the variety in which the perforation is in the free bowel. Here, also, adhesions presumably form as the ulcer approaches the serosa, but inasmuch as they are attached to a movable part of the bowel they cannot be relied upon to hold, and extravasation usually soon takes place. It is with perforation of the ileum that we are chiefly concerned, and in looking for information upon this subject we are hampered because we find commingled in the statistician's tables, two very different and widely separate conditions, one, the results of operation for typhoid perforation, the other, the results of operation for general peritonitis following typhoid perforation.

The mortality following operation for general septic peritonitis, due to extravasation of intestinal contents, is necessarily high. Could these cases be excluded from the tables we should find that operative interference in typhoid perforation is associated with a moderately low mortality.

A consideration of our cases, and of some heretofore reported, emphasizes the necessity of early operation upon the first symptoms of perforation, or possibly upon recognizable pre-perforative symptoms, without waiting for the usual signs of peritonitis. It is far better to operate early, needlessly if it so eventuate, rather than to wait until symptoms of peritonitis appear and actually demonstrate a perforation by its dread and practically inoperable sequel of general septic infection of the peritoneal cavity.

Any abdominal symptoms occurring in the course of the fever are as urgent indications for a surgical consultation as is the appearance of pain and tenderness in the right iliac fossa under all occasions, and only when this is fully realized will the mortality of these cases approach the low percentage reached in operations for acute perforative appendicitis or perforating gunshot wounds of the abdomen. Delay in these two latter conditions is no longer thought of, and equally prompt intervention on the first abdominal symptoms in the course of typhoid, without waiting for actual evidence of peritonitis, will similarly reduce its high death-rate. It is hard to understand Dr. Keen's advocating delay until symptoms of shock have passed away and his preference of the second twelve hours for operating, when one appreciates that extravasation, perhaps of virulent organisms, is with all probability continually taking place while we are waiting.

There are of course certain cases, of which Dr. Osler* makes mention, in which perforation gives rise to no signs whatever as the patients are desperately ill and the local features are masked by the severity of the toxæmia. The diagnosis is usually made at such times on the autopsy table. Hospital cases, however, are usually carefully watched and some symptoms almost invariably should give warning of the complication, if not before, certainly at the time of perforation.

The figures, however, as they are given, including cases of all descriptions, even those condemned before they reach the operating room, present a comparatively low mortality.

Westfall's statistics (1898) given by Keen† are the most recent, and show 19.36 per cent. of recoveries in 83 collected cases. Those of Finney‡ (1897) include forty-five fairly authentic cases, with eleven recoveries, making his statistics somewhat better with 26.22 per cent. of recoveries. Monad and Vanverts§ consider the mortality to be much greater, namely 88 per cent., contrasted with a supposed 95 per cent. of deaths in unoperated perforations. With this small margin, however, they strongly recommend operation.

It is probable that the last figures more nearly represent the truth, as there are presumably many cases lost from tardy operations, which are never reported, and in the more favorable statistics given above there are doubtless some cases included which are of questionable typhoid origin.

Only in recent years has it become possible by bacteriological examination and by the serum reaction to conclusively demonstrate the nature of certain fevers. The writer recently operated on a perforated appendix associated with a general fibrino-purulent peritonitis due to a colon infection in a patient who subsequently had for three weeks a typical typhoid

* **Fitz**, R. H.: Intestinal Perforation in Typhoid Fever: Its Progress and Treatment. Trans. of the Assoc. of Am. Phys., Vol. VI, p. 200, 1891.

* **Osler**: Practice of Medicine, 3d edition, 1898, p. 26.

† **Surgical Complications and Sequels of Typhoid Fever**, 1898, p. 234.

‡ **Finney, J. M. T.**: The Surgical Treatment of Perforating Typhoid Ulcer. The Annals of Surgery, March, 1897.

§ **Monad, Ch. et J. Vanverts**: Du traitement chirurgical des péritonites par perforation dans la fièvre typhoide. Revue de Chirurgie. T. XVII, 1897, p 169.

chart and a general typhoidal appearance without leucocytosis, and with no abdominal symptoms. This would undoubtably in former years have been considered an appendicular typhoid perforation. Only after persistent negative results with the Widal reaction could we believe the case to be non-typhoidal.*

Another case, which would certainly have been considered a perforation in an ambulatory typhoid had not careful microscopical and bacteriological study been made of the tissues, is as follows:

The patient, Fred. H., aged 26, having been discharged from the work-house the day previously, entered the hospital January 25, 1897, after 12 hours of acute abdominal distress. He had all the symptoms of general peritonitis, and at operation a single perforation was found in the ileum the size of a five-cent piece and about ten inches above the ileo-cæcal valve. He died 6 hours later, and the necropsy revealed an acute splenic tumor, parenchymatous degeneration of the liver and kidneys, but no other intestinal lesions characteristic of typhoid. There were no focal necroses in the liver, and the bacillus typhosus was nowhere obtained in cultures.

This case of perforation of the ileum, evidently not typhoid, presents such similarity to the notable one of Miculicz, which is usually admitted to have been of typhoid origin, that I cannot but believe the latter also was due to a perforation not resulting from typhoid fever, though its exact nature must remain uncertain. Doubtless many others of the tabulated perforation cases would likewise be discarded as "not typhoid" could they be scrutinized in the light of more recent and positive methods of diagnosis.

Diagnosis.—The question of early diagnosis of typhoid perforation is unfortunately but little touched upon in the recent monographs upon the subject, which give little more than a stereotyped picture of pain, collapse, vomiting and abdominal tenderness, a symptom complex which is enough of course in ordinary cases to assure one of the condition. We have seen illustrated by the above cases, however, that this picture is but rarely complete, and the difficulties in the way of the recognition of perforation are frequently so great that it may be overlooked entirely. Two of them also show that other conditions may give the characteristics typical of perforation when this complication has not occurred.

The complete symptomatology is usually given as follows. During the course of the fever, usually in the third week of a severe attack, most often in male adults there appears, with sudden onset, abdominal pain usually in the right side, associated with more or less tenderness and rigidity. Vomiting follows with more or less irregularity. The onset may be associated with a chill and pyrexia, or with cold extremities, collapse and a drop in temperature often of several degrees. The pulse becomes small and wiry. Leucocytosis is supposed to make its appearance, and soon more or less abdominal distension sets in with increase of vomiting, shifting dulness in the flanks, obliteration of liver dulness, a gradual return of pyrexia if there has been a fall, with rapid feeble pulse, restlessness and thirst, all indicative of general peritonitis, with death supervening in 24 to 48 hours.

Of these symptoms, especially those associated with the onset, a few remarks will be made.

Abdominal pain and tenderness.—It is a well-recognized fact that the character of the symptoms in typhoid fever seems to vary in different years within considerable limits. An unusually large number of the cases which have been treated in our medical wards this fall have had abdominal pain and tenderness which have frequently been associated with diarrhœa. This has been so pronounced a feature that several cases have been seen in surgical consultation for symptoms which have subsequently disappeared. A sudden acute onset of increased abdominal pain is an all important symptom which unfortunately may be absent, or owing to a patient's stupor be overlooked. Any complaint of pain, however, of less abrupt onset, associated with tenderness, must arouse the greatest suspicion on the part of the attendant. I cannot but believe that the condition which has been spoken of above as a pre-perforative stage of ulceration often exists. A little localized inflammation of the serosa, with or without the passage of microorganisms and leading to a slight adhesive peritonitis, usually of omentum, can give rise to these symptoms and produce an associated slight leucocytosis. This is precisely analogous to what occurs in the pre-perforative stage of appendicitis which, however, is of less urgent nature because in the case of the appendix which is fixed and does not move about freely in the general cavity, as do the coils of ileum, the adhesions are less likely to be dislocated and a general peritonitis, which would result from this separation, is avoided. This is as true of appendicular perforations in typhoid as of those occurring at other times. I believe that this pre-perforative stage may be frequently recognized as in Case III reported above. Doubtless in some of the successful cases of operation for perforative peritonitis such a pre-perforative stage has been met with. This was notably so in Dr. J. B. Murphy's case,* where no perforation was found, but merely a local inflammatory reaction about one threatening ulcer. Several of the other successful cases illustrate a pre-extravasation stage where adhesions had reinforced the serosa before it gave way entirely and had temporarily prevented extravasation. Among such cases may be mentioned Watson's,† Bogart's‡ and our first case at the third operation.

Under rare conditions when the adhesions are firm, which, for the reasons mentioned above, is more likely to occur when the appendix is the seat of threatened perforation, the base of the ulcer may completely penetrate the bowel and yet the general cavity be protected. A local abscess may result, or the adhesions may floor the ulcer and subsequent healing take place over them so that recovery follows without operative intervention.

This is the usual explanation of recovery following symptoms of perforation, in cases which have not been subjected to operation. In the case of Buhl,§ quoted by Fitz, at an autopsy

* This case subsequently came to autopsy and a tuberculous enteritis was found. The appendicular perforation was probably through a tuberculous ulcer.

* Westcott's table, Case No. 41. Keen.

† Watson: Boston Med. and Surg. Journ., Vol. CXXXIV, 1896.

‡ Bogart: Op. cit.

§ Zeitschr. f. rat. Med., 1857, N. F. VIII, S. 12.

following death from hæmorrhage, a pre-existing perforation was found to have been closed by omentum. This was twenty-three days after the occurrence of symptoms of perforation. Dr. Hare* of Brisbane, says: "At present it is an open question whether the treatment should be medical or surgical; whether indeed laparotomy is justifiable." He reports an interesting case in which symptoms of perforation had occurred. The patient subsequently died, some time later, with dysenteric symptoms, and the ileum was found surrounded by adhesions, which were especially dense at the point corresponding to a supposed perforation. I do not think, however, that this case is at all conclusive. A threatened perforation with perhaps the escape of some organisms through an intact serosa, which Dr. Welch has proved to be possible, would have accounted for the localized peritonitis. Had the perforation been complete, doubtless the adhesions would not have long sufficed to confine the extravasation. In his second case of supposed recovery after perforation an operation, had it been offered in the first hours of symptoms, would with greater probability have insured success. Mr. Gairdner's† interesting cases also would show that a fatal peritonitis without an absolutely complete perforation may take place. He reports five such instances.

The protection by adhesions in this way is too precarious a thing to be relied upon, and that they should hold for any length of time is something which can never be anticipated. The recognition of this pre-perforative stage I would emphasize as all important.‡

This is the period in which, if possible, an operation should be performed, and as it may endure but a short time, the opportunity should be immediately seized. Such a condition existed in Bogart's case§ in which he found a perforation of the ileum closed and the ulcer floored by the adherent tip of the appendix. He speaks of the presence of sero-purulent fluid in the general cavity which doubtless was free from organisms as it was in our Case I, which was operated upon before extravasation of intestinal contents had taken place. An opportunity of operating in this stage was unhappily neglected in Case III.

An analogous pre-perforative stage was recognizable in the following case, one of dysentery, upon which the writer recently operated, though too late. The patient had been in the medical wards for some days with a severe amœbic dysentery. He developed considerable abdominal pain, tenderness and leucocytosis, with some rigidity of the parietes. Several days later, while having a rectal irrigation, sudden evidence of perforation and extravasation occurred with acute pain and collapse. At the operation, three hours later, his abdomen was full of fæces, which were pouring from a large opening in the sigmoid flexure. The autopsy revealed an extraordinary degree of ulceration of the colon with a complete loss of substance in the bowel in several places, but all of these ulcerated areas, except the one found at the operation, were completely floored by protecting omental adhesions.

As in the three typhoid cases reported above, here too was a distinctly recognizable pre-perforation or pre-extravasation stage of intestinal ulceration which demanded operative relief before final signs of perforation with extravasation had rendered the chances of giving it most desperate.

Undoubtedly, signs exist which are often considered trivial, but which may aid us in anticipating a final perforation by indicating early laparotomy.

Temperature and Pulse.—A pronounced drop in temperature is a not infrequent symptom, associated with the onset of the perforation. It must, however, be clearly distinguished from the great fall in surface temperature, which is often pronounced and gives rise to the cold and clammy extremities so characteristic of the collapse of onset. This latter condition, however, may be associated with a rise of central temperature. In some of the cases cited by Fitz this collapse was the only symptom indicative of perforation.

The sudden fall of the central temperature, when it occurs, is such a pronounced feature that more importance has been ascribed to it than it deserves. Dieulafoy* considered it an almost infallible sign. He says: "La perforation intestinale, au cours de la fièvre typhoide se traduit dans la très grande majorité des cas, par une *chute brusque de la température.*" He thinks the appendicular attacks occurring in the course of the fever show, on the contrary, a rise in temperature, and may thus be distinguished. Lereboullet,† however, takes exception to this statement in a thorough discussion, and believes it to be exceptional. Gesselewitsch and Wanach‡ also emphatically assert that many cases are accompanied by a rise in temperature. One can merely state that when present it is a characteristic symptom, but that it may be absent. It also, of course, frequently occurs in other conditions such as hæmorrhage, and our Case I further illustrates its unreliability as there was no drop with the perforation, but a pronounced fall with the obstruction and after each operation. It may possibly be a means of distinguishing, as Dieulafoy suggested, between a perforation with extravasation into the free cavity and one protected by adhesions giving merely a local inflammatory reaction.

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* The Cold Bath Treatment of Typhoid Fever, 1898, p. 178.

† Gairdner: Peritonitis in Enteric Fever. The Glasgow Med. Journal, Vol. XLVI, p. 114, Feby., 1897.

‡ Under "pre-perforative stage" let it be understood that the whole period is included between the first involvement of the serosa with the customary formation of adhesions at that point, until these adhesions, which may for a time constitute the floor of the ulcer after the serosa has given way, have themselves become broken down and general extravasation has taken place. This period as in perforating appendicitis may last a longer or shorter time and is associated with pain and tenderness and a possible rise in leucocytosis owing to the localized peritonitis.

§ Bogart, J. Bion: loc. cit.

* Dieulafoy: De l'intervention chirurgicale dans les péritonites de la fièvre typhoïde. Bull. de l'Académie de Médicine. Oct. 27, 1896.

† Lereboullet: Sur le diagnostic et le traitement des perforations intestinales dans la fièvre typhoïde. Bull. de l'Acad. de Méd. Nov. 3, 1896.

‡ Gesselewitsch and Wanach: Die Perforations Peritonitis beim Abdominal Typhus und ihre operative Behandlung. Mitteilungen aus den Grenzgebieten der Medizin und Chirurgie, Bd. I, H. 1 und 2, 1898.

The disparity between pulse and temperature may be a marked feature, the former being small and rapid during the drop in temperature. The respiration likewise is apt to be more rapid and shallow with less marked abdominal movements.

Of symtoms other than those associated with a *threatening* perforation or with its immediate occurrence little need be said. A chill sometimes occurs, but more often with the circumscribed and appendicular varieties, when, too, the temperature is more apt to rise. Vomiting is an important symptom when present, but its frequent absence makes it an unreliable one. The acoustic phenomenon of Levaschoff, a sound caused by the passage of gas through the perforation with each descent of the diaphragm, has not been generally confirmed. Later signs, such as shifting dulness from free fluid, distension, obliteration of liver dulness and other indications of abundant extravasation of gas and fæces, such as were present in Case II, make the diagnosis of a long standing perforation as easy as its prognosis is unfavorable. Even many of these stereotyped indications of general peritonitis may be misleading. In Case III shifting dulness was a marked feature, and yet but little free fluid was present. Similarly a distended colon may cause partial obliteration of liver dulness, but even with perforation and extravasation too small an amount of gas may escape to produce it.

Leucocytosis.—Of great interest and of great diagnostic importance in these cases would seem to be the presence or absence of leucocytosis.

The final interpretation, however, to be given to this symptom is far from being made. Dr. Finney says: "Of all the so-called diagnostic signs of perforating typhoid ulcer most reliance is to be placed upon the development of an attack of severe, continued abdominal pain, coupled with nausea and vomiting, and at the same time a marked increase in the number of white blood corpuscles." We have seen, however, in some of our cases a fall and not an increase in leucocytes, which must receive consideration.

The fact that "there is not only no increase in the proportion of colorless corpuscles during the fever, but that on the contrary there is rather a tendency toward a diminution in number at the height of the disease," was emphasized by Thayer* in 1892. The occurrence of leucocytosis therefore is quite properly in most cases supposed to be coincident with the presence of some septic complication other than the surface ulcerations of the intestinal tract. Cabot† is inclined to the belief that in all the cases in which leucocytosis exists constantly, some complication really *is* present though it may be unrecognized. He cites two cases with a leucocytosis of 24,000 and 18,000 respectively, occurring at the time of perforation. He further states: "It occasionally happens in very exhausted patients that complications fail to produce any leucocytosis, the patient (as in some cases of pneumonia or purulent peritonitis) being unable to react against the infection" (p. 170). This statement, I think, needs some qualification.

Using the cases above reported, in all of which careful leucocyte counts were made, we are confronted by quite a different picture. In Case II the complete absence of leucocytosis was the unfortunate cause of a deferred operation. I doubt not, however, that a leucocytosis, which subsequently disappeared, was present at the onset of the peritonitis.

In Case I there was an early and recognized leucocytosis appearing, however, before any signs of general peritonitis had developed; and in the peritoneal fluid comparatively few white cells were present and no micro-organisms.

In Case III, a preceding leucocytosis associated with abdominal pain and tenderness, which, as has been stated, was probably indicative of a mild local peritonitis about the extensively ulcerated bowel, was completely wiped out concomitant with the occurrence of general peritonitis and the appearance of great numbers of leucocytes in the extravasated peritoneal fluid.

In the case of dysenteric perforation, mentioned above, the leucocytosis had previously been constantly high. A few days before the perforation it was 47,000. At the time of perforation it was 41,000. An hour later it had fallen to 30,000 and at the time of operation it was 27,000, a drop of 20,000 in three hours. At the operation the lower bowel was matted together with adherent omentum, this local inflammatory process doubtless being the cause of the preceding leucocytosis. The general cavity was full of fæcal and purulent fluid, in which were great numbers of polymorphonuclear leucocytes, eosinophiles and mononuclears in about the proportions found in the blood. Many of these cells were crowded with organisms and disintegrating. There is but one natural conclusion to be made from this sudden diminution of the number of white cells in the peripheral circulation coupled with their appearance in the peritoneal exudate.

Similarly in appendicitis the writer has frequently seen, after a high percentage of leucocytes present during the acute stage, a drop in their number occurring in association with the onset of peritonitis, as characteristic as that which occurs with the subsidence of the acute attack and recovery.

In Cabot's table XXI of counts made in general peritonitis, there are 4 without leucocytosis, the numbers varying between 4600 and 6000. Of those with leucocytosis, as well as in his cited dysenteric case with 24,000 leucocytes there is no recognition of a possible fall in number such as occurred in the cases cited above after the onset of general peritonitis.

Cabot* says: "A steadily increasing leucocytosis is always a bad sign and should never be disregarded even when other bad symptoms are absent." I would add that a decreasing leucocytosis may be a much worse sign, and should never be disregarded. This is especially of importance in those typhoid cases in which the "other bad symptoms" are difficult to estimate on account of the dull condition of the patient.

From these data on leucocytosis the following conclusions may be drawn:

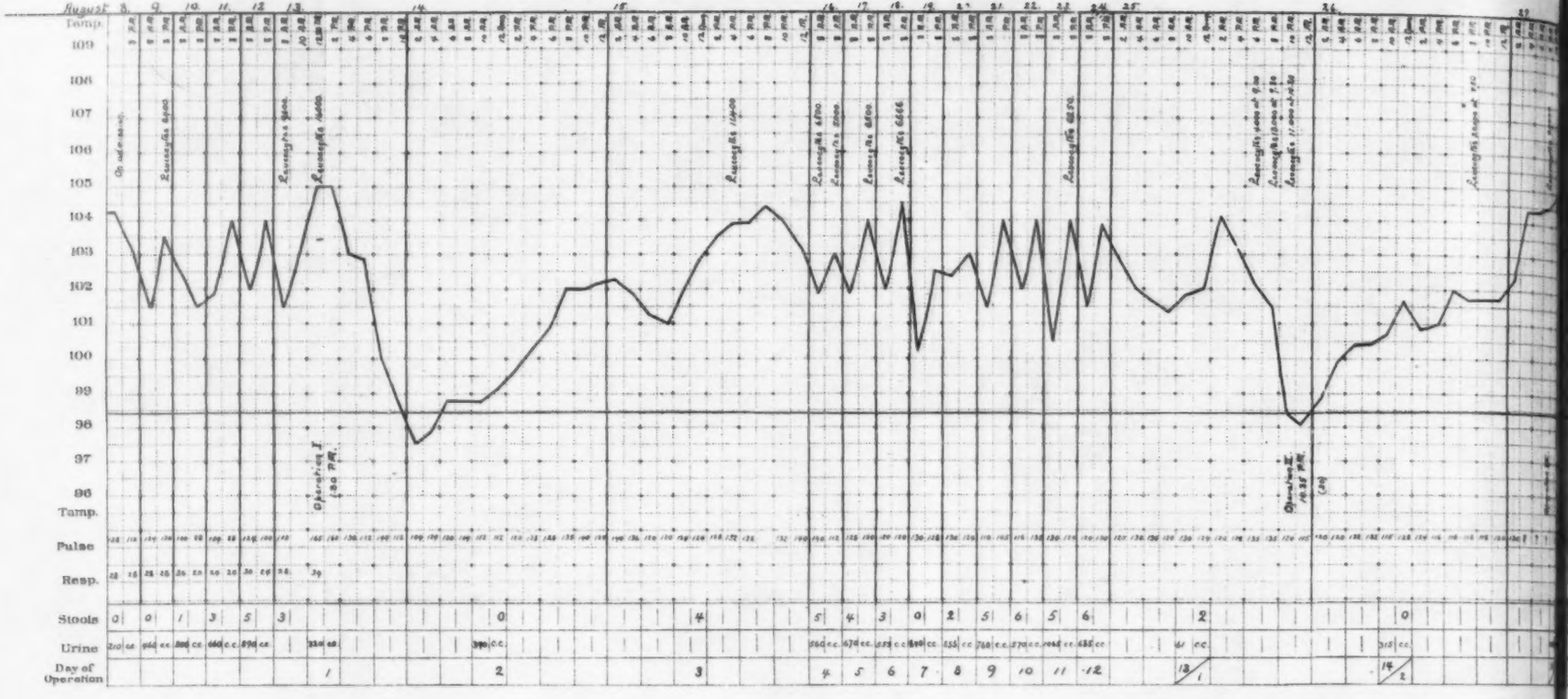
1. The appearance of leucocytosis in the course of typhoid fever points toward some inflammatory complications in its early stage.

* Thayer, W. S.: Two cases of Post Typhoid Anaemia. With Remarks on the Value of Examinations of the Blood in Typhoid Fever. Johns Hopkins Hospital Reports, Vol. IV, No. 1, p. 88.

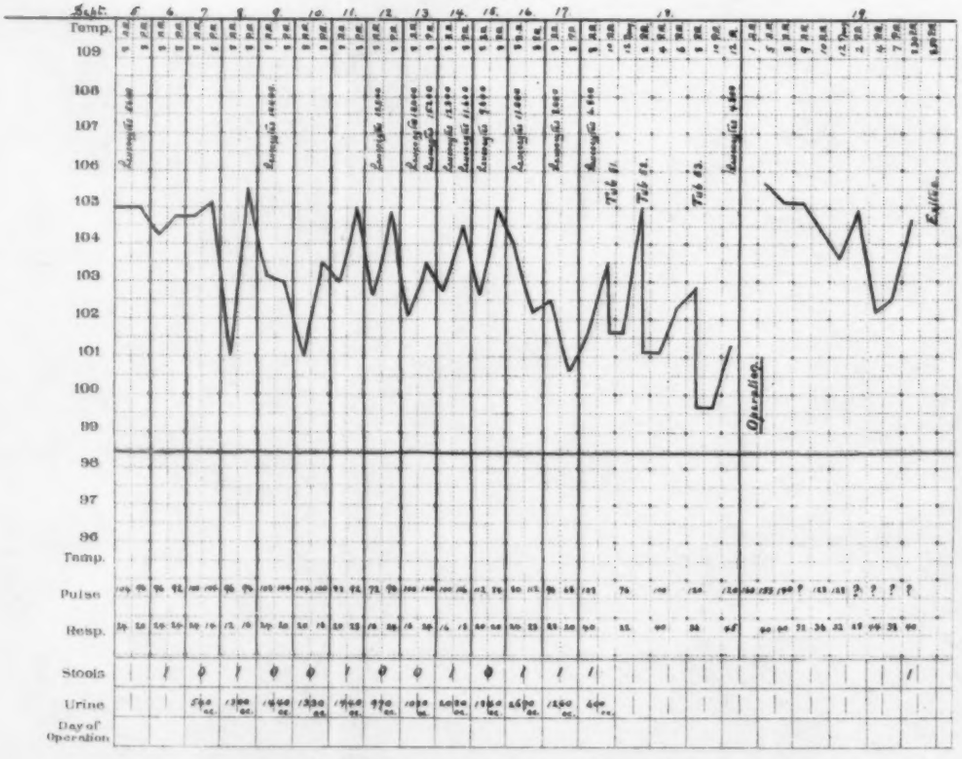
† Cabot, R. C.: Clinical Examination of the Blood, p. 168, 1897.

* Op. cit., p. 197.

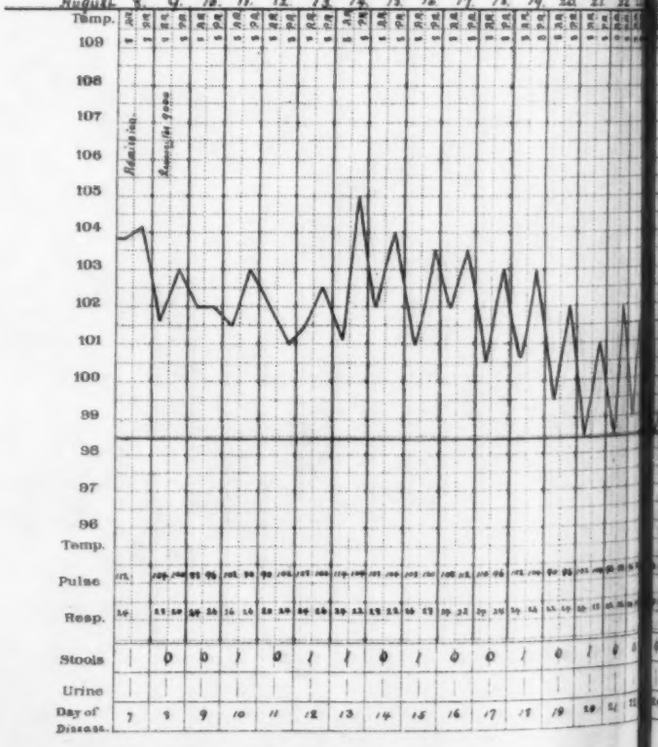
CASE I.



CASE III.



The complete chart showing the tubs is only given on the 18th inst.



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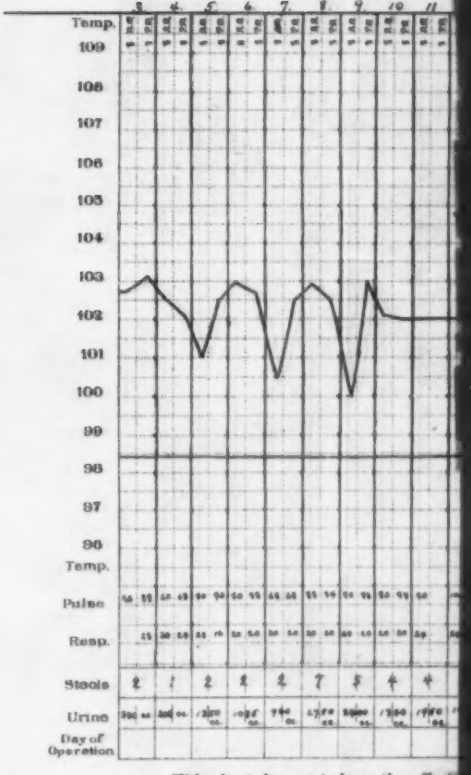

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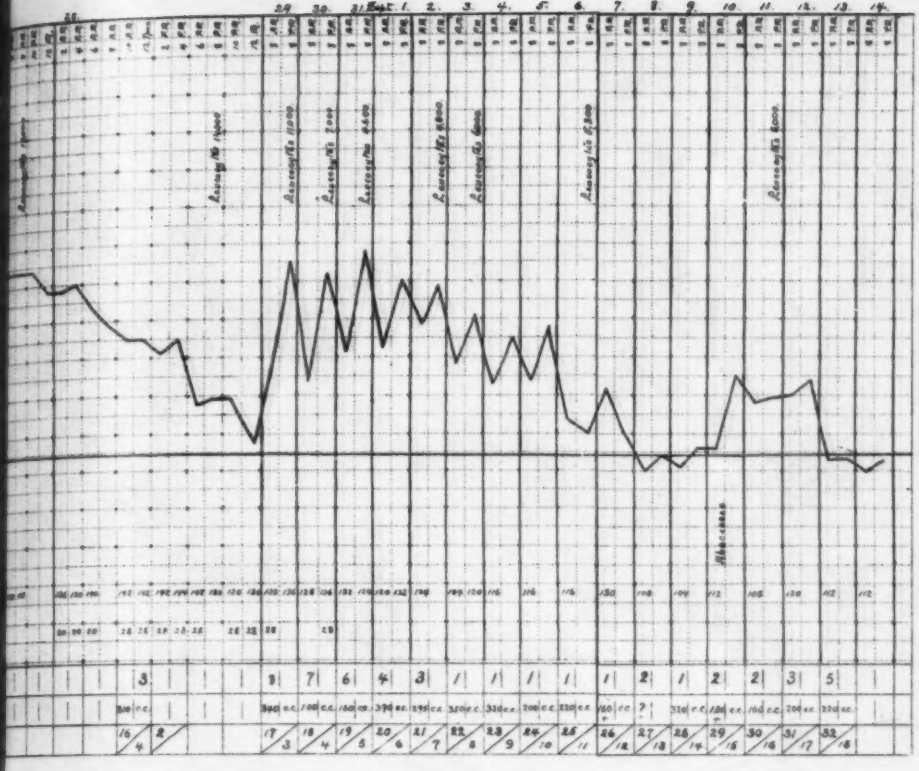

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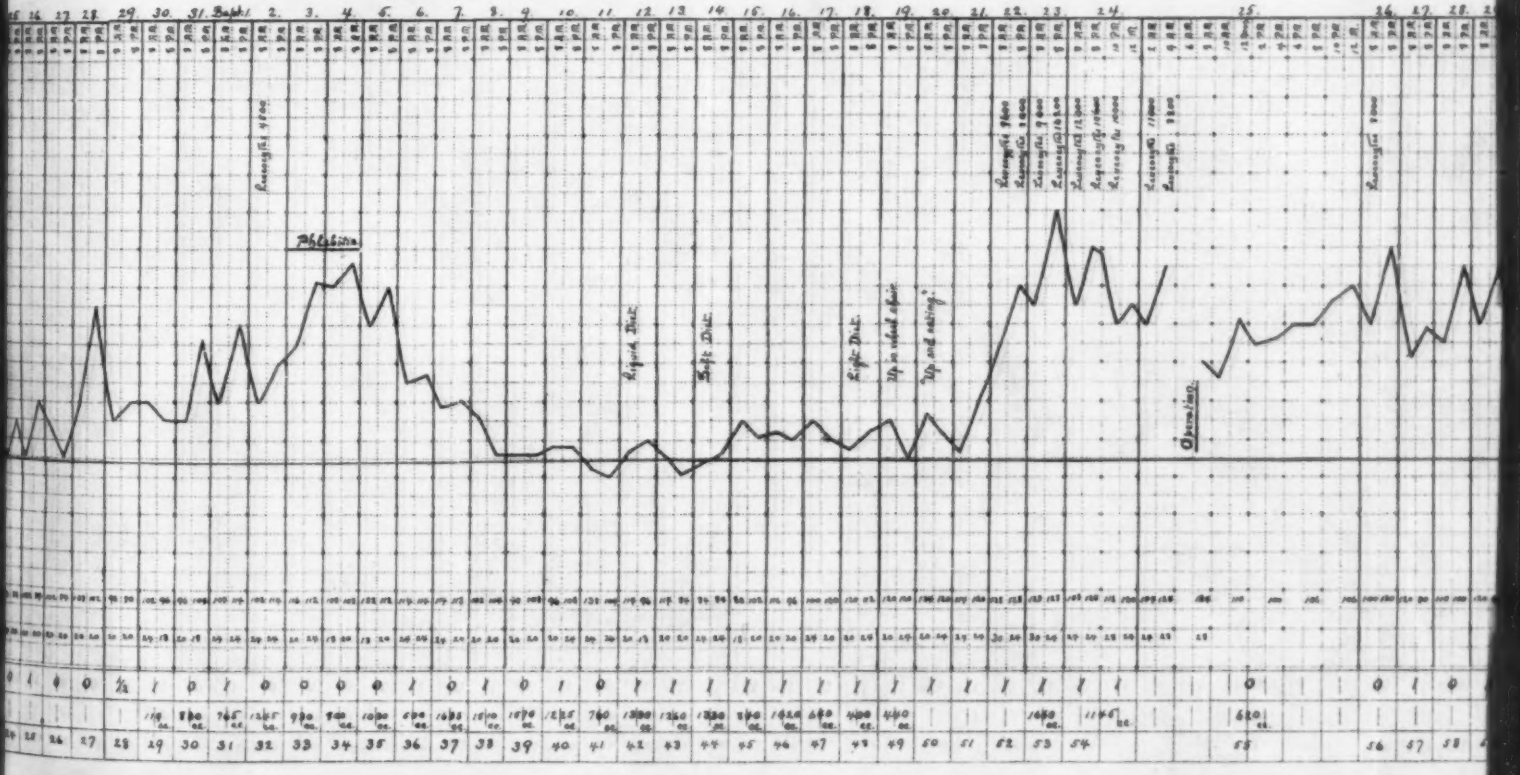
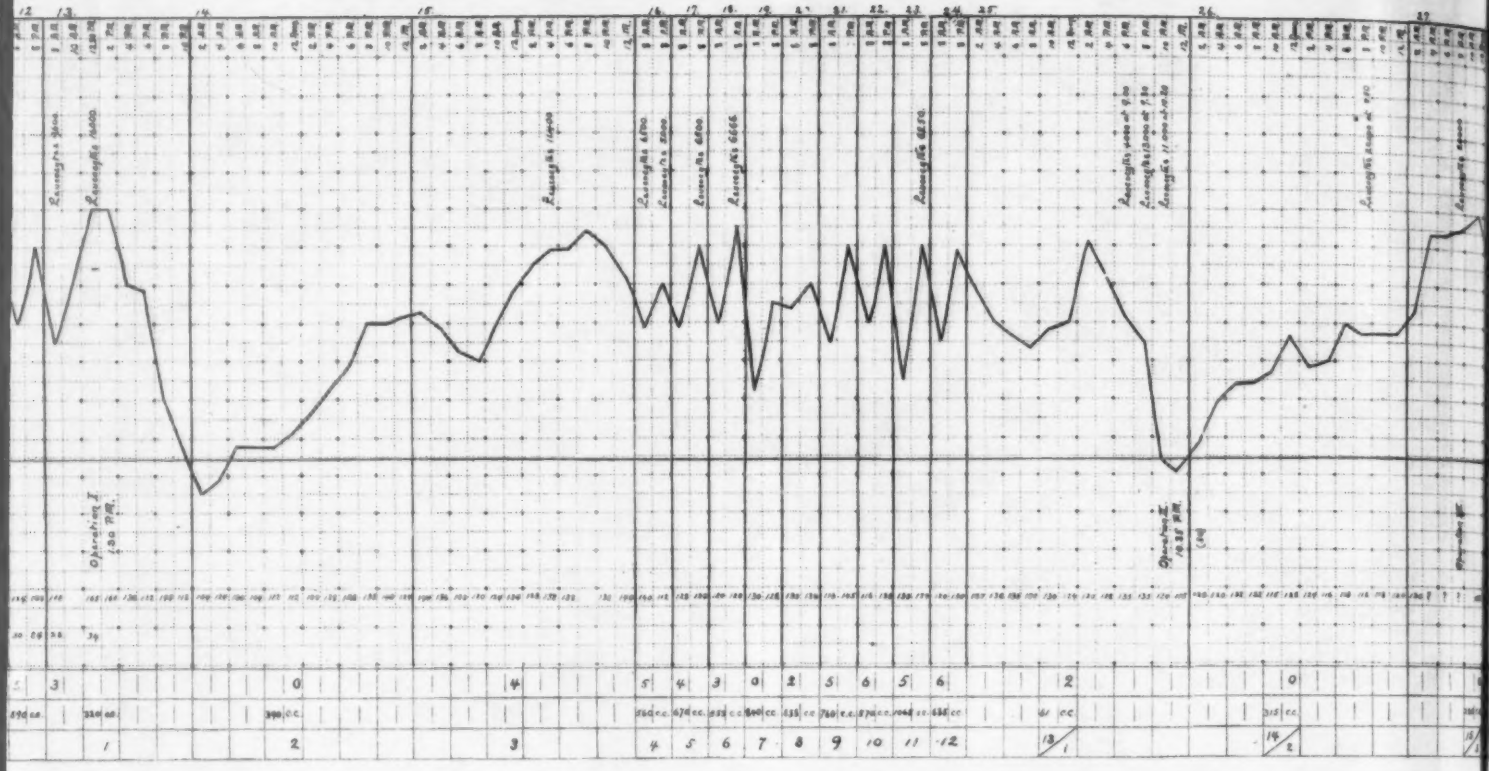
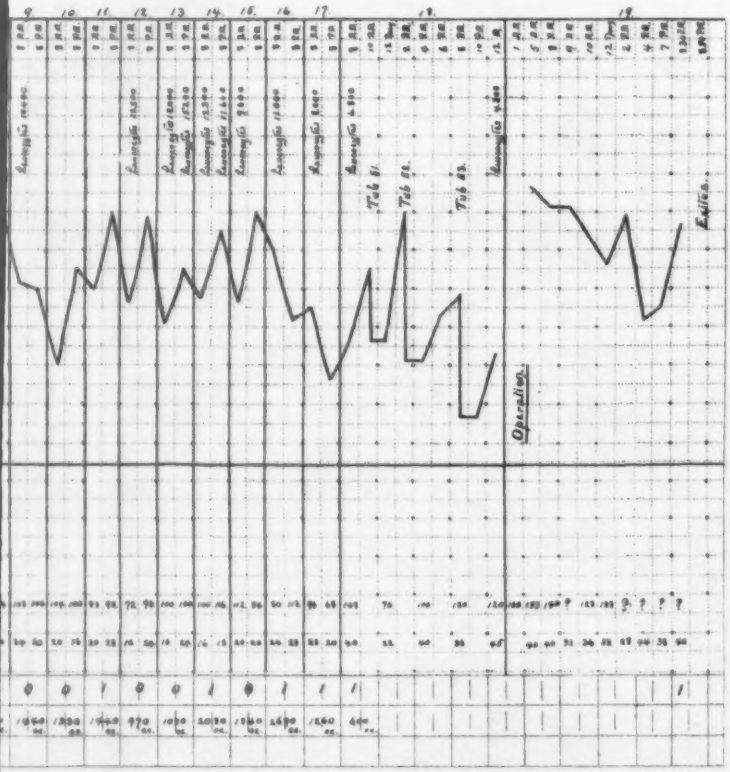
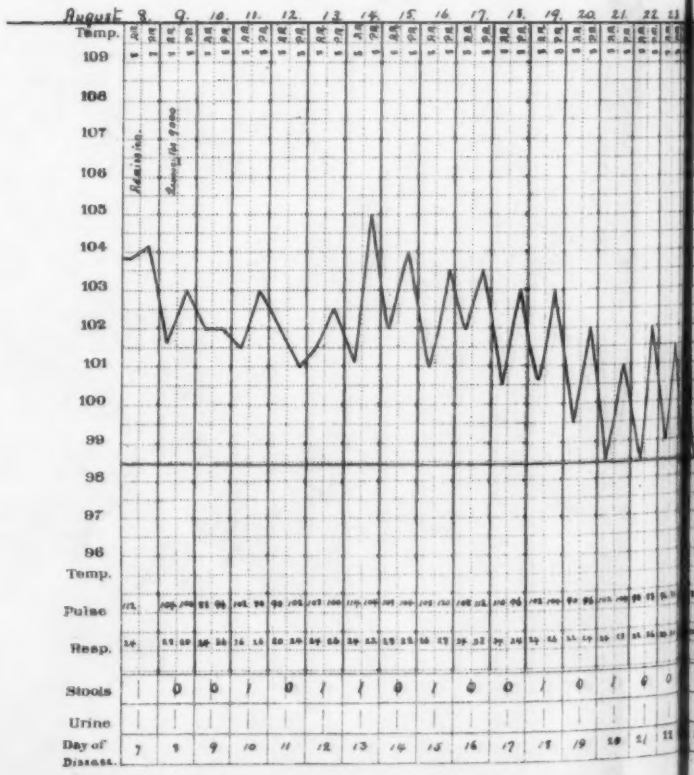


CASE I.



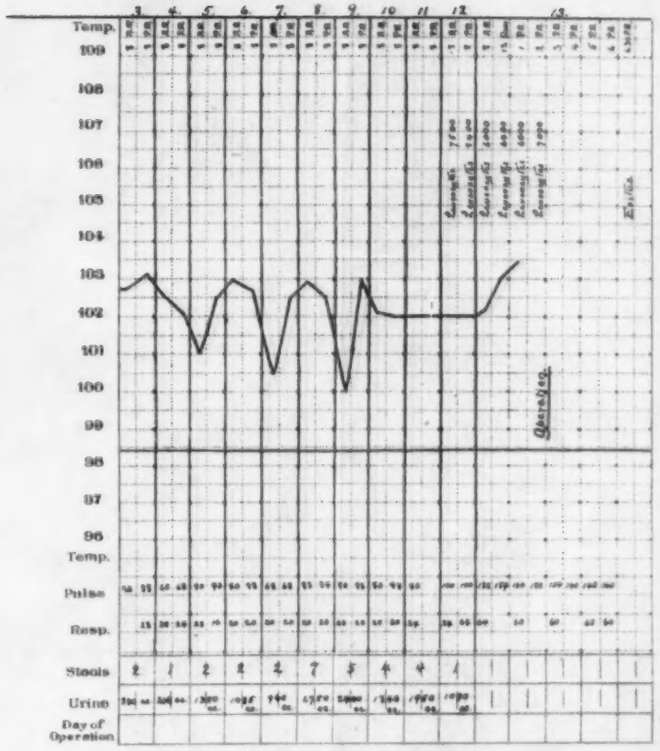
CASE III.





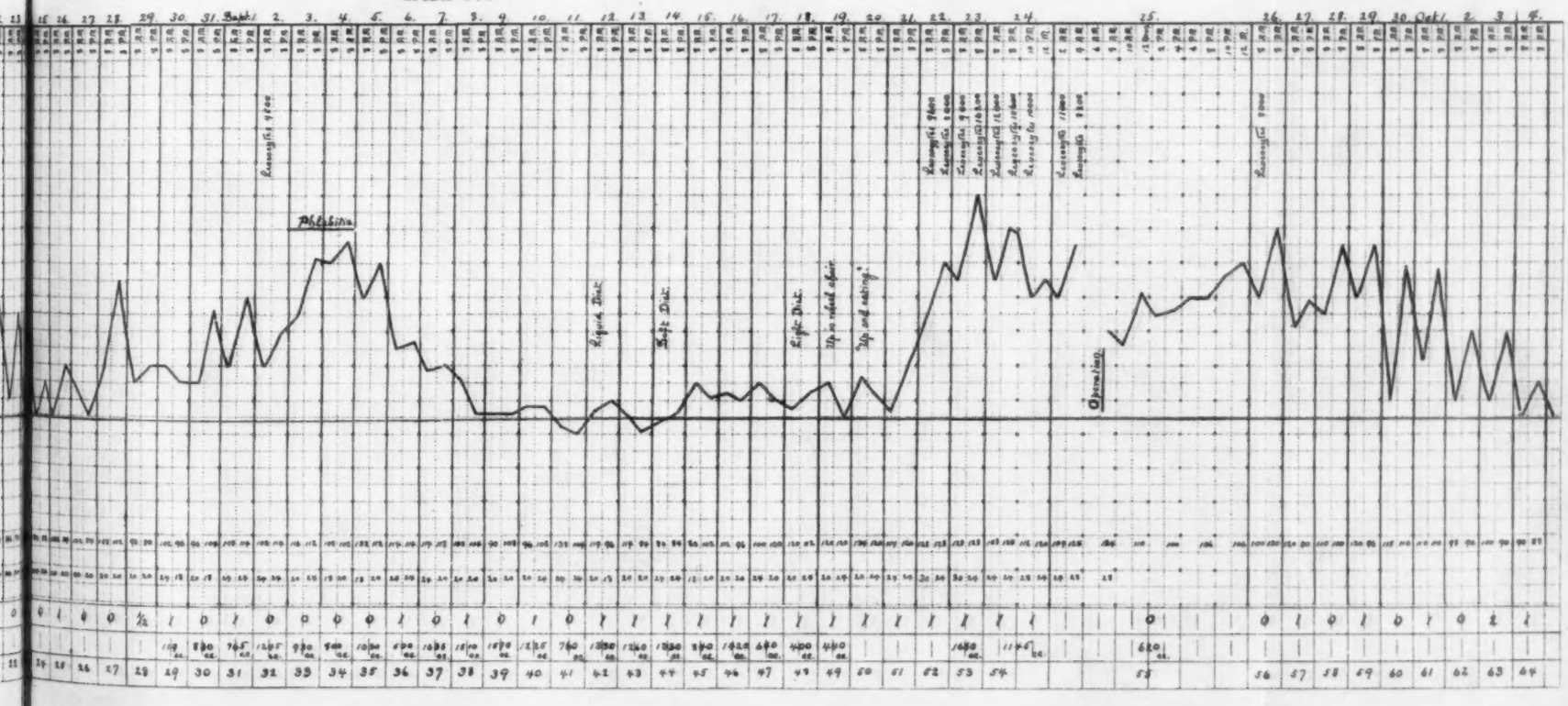
The complete chart showing the tubs is only given on the 18th inst.

CASE II.



This chart does not show the effect of the tubs.

CASE IV.



2. If this complication be a peritonitis and remain localized, associated possibly with a pre-perforative stage of ulceration (cf. Case III) or with a circumscribed slowly-forming peritonitis after perforation, it may be and usually is signalized by an increase of leucocytes in the peripheral circulation.

3. If, however, a general septic peritonitis follow, the leucocytosis may be but transitory and overlooked, as it disappears concomitantly with the great outpouring of leucocytes into the general cavity.

The various forms of operative procedure advocated in these cases it is not the object of this paper to discuss. They are fully set forth in the recent monographs on the subject by Finney, Gesselewitsch, McCosh, Keen, and Farrar Cobb.*

CONCLUSIONS.

The diagnosis of intestinal perforation in typhoid fever may present many difficulties. No abdominal symptoms either subjective or objective occurring in the course of the fever should be regarded as trivial, and a sudden change of any sort in the patient's condition should lead first of all to the suspicion of this most serious complication. A distinction should be drawn between the two varieties of perforation, the appendicular and those occurring in the free bowel, as their symptoms, course and prognosis vary considerably. Many cases, however, even those of perforation from the free bowel, present what may be recognized as a pre-perforative stage which in some cases calls for a laparotomy in anticipation of a complete perforation with extravasation. The presence of leucocytosis is not an infallible sign of perforation as it may disappear with the onset of general peritonitis. It is most valuable in this anticipatory stage.

When the diagnosis is made operation is indicated, whatever the condition of the patient. As Abbe's case exemplifies, no case may be too late. A precocious exploration from an error in diagnosis is not followed by untoward consequences such as must invariably be expected after a neglected and tardy one.

Our present knowledge amply corroborates the statement of Miculicz made at Madgeburg in 1884: "If suspicious of a perforation one should not wait for an exact diagnosis and for peritonitis to reach a pronounced degree, but on the contrary one should immediately proceed to an exploratory operation, which in any case is free from danger."

DISCUSSION.

DR. FINNEY.—There are a number of points in Dr. Cushing's most interesting paper to which I should like to call attention. Some of the points he has made are new, and I think a distinct addition to our knowledge of the subject. This is a subject in which I have been, personally, very much interested and have recently had occasion to go over the literature of the operation pretty thoroughly. I have myself operated upon four cases with one recovery; the other three were practically moribund at the time of operation. They were forlorn hopes, but as Dr. Abbey's case recovered, and since we have nothing to lose by operating in such cases, we may occasionally gain something. Next to Dr. Cushing's case, which must take the palm for having recovered under the greatest difficulties, I think my case a good second. Dr. Cushing's patient had three successive laparotomies, and subsequently recovered. My case had but one operation, but he had almost every complication that a typhoid patient can have.

In regard to the question of leucocytosis, we expressed the hope a year or two ago (the suggestion being first made by Dr. Thayer) for much of diagnostic value in the blood count of these doubtful cases. It has been of some value, but just how much remains to be seen. Dr. Cushing mentions the fact that in one case the increased number of leucocytes suggested perforation, and in another the rapid decrease in leucocytes was suggestive, if not of perforation, at least of a critical condition of the patient. The question of the value then of the blood count and its diagnostic significance remains to be determined.

In Dr. Cushing's third case I find the only one, in about a hundred which I have collected, occurring in a negro, and he died.

The question of a pre-perforative stage, as Dr. Cushing has called it, is very interesting and important if it can be really differentiated. It is a point which will be further investigated with a great deal of interest. As to statistics, it depends entirely upon how many doubtful cases one admits into one's series. In our previous report we found 26.2 per cent. of recoveries. Then Dr. Keene's very exhaustive work appeared in which he reduced the percentage to 19.6 per cent., but since then there have been a number of cases that will, I am glad to say, bring up the percentage somewhat. I have not a complete history of some of these cases as yet, but I think it will average between 20 per cent. and 25 per cent. The distinction which Dr. Cushing has drawn between operating for the relief of a perforating typhoid ulcer, and a general septic peritonitis following such a perforation, is a very good one.

After all, early operation is the main point in this as in many other operations. If we can get these cases early enough we shall have a much larger percentage of cures, and there, as Dr. Osler has suggested, the physician and surgeon must work together and try in every way to increase our diagnostic ability. So far as the technique of the operation is concerned, I believe there is not much to be gained, and we have reached the point in dealing with these cases where we haven't much to hope for in that direction, but must turn in the direction of an earlier diagnosis for any marked improvement in our statistics.

DR. OSLER.—I think Dr. Cushing is to be congratulated upon his excellent results. Certainly to save one case in three of perforation is much more than we can do on the medical side, for they all die with us, except a few cases of appendical perforation.

Dr. Cushing has pointed to the difficulty of diagnosis, and I do not know of any more difficult problem than to determine in some cases the existence of perforative peritonitis. The local symptoms may predominate, and I would ask whether in the case of the young girl the general symptoms of collapse, change in countenance, pulse, etc., were present or not?

There are many other points in connection with this question that I would like to discuss, but the programme is a long one and there will probably be other speakers. I would, however, refer to one point mentioned, the great importance, when the autumnal crop of typhoid cases is in hospital, that the house surgeon and house physicians should often make rounds together.

*Farrar Cobb: Septic Peritonitis and its Surgical Treatment. Boston Medical and Surgical Journal, Sept. 8, 1898.

SPONTANEOUS HÆMORRHAGIC SEPTICÆMIA IN A GUINEA-PIG, CAUSED BY A BACILLUS.

By George H. Weaver, M. D., *Chicago.*

(*From the Pathological Laboratory of Rush Medical College.*)

The bacillus here described was obtained from a large female guinea-pig, which died suddenly without apparent cause in the animal room attached to the pathological laboratory of Rush Medical College.

Upon examining the body, the condition of the uterus first attracted attention. The right cornu was swollen to a diameter of 1 to 1.5 cm. for 5 cm. of its length. The covering of peritoneum exhibited marked vascular injection, but no exudate. The opposite cornu was of the normal size and appearance of the unimpregnated uterus. On opening the uterus from in front the cavity was found empty. The mucous membrane of the right cornu presented numerous scattered hæmorrhagic areas, which became confluent in front. On the posterior surface, about midway between the bifurcation and apex of the horn, was an area $1\frac{1}{2} \times 2$ mm. in diameter, slightly elevated, firm, and of a dirty blackish-brown color.

The spleen was enlarged and soft; the kidneys and liver pale. The heart was distended with blood. The lower lobe of one lung was firm, airless, and of a dark red color. In the opposite lung was a smaller area of firm consistence, of a deep red color, slightly depressed below the surface. On section the solidified portions were yellowish in color, and airless.

Cultures upon agar-agar, from the uterine wall, spleen, kidneys, heart-blood, and pneumonic area gave an abundant growth of the bacillus to be described below. Portions of the organs, hardened in alcohol, and stained with hæmatoxylin and eosin, and with Loeffler's methylene-blue, were examined with the following results:

Uterus.—The mucous tissue is largely destroyed in the right cornu at the site of the necrotic area. The superficial layer consists of necrotic cells, some with fragmented nuclei, some containing blood-pigment. In some places are areas of hæmorrhage with well-preserved blood-cells, the infiltrated tissue staining poorly. The tissues beneath the serosa are much swollen, and the capillaries here much dilated. The muscular coat is much thickened from enlargement of the fibres and separation of them by fluid exudate. There is no round-cell infiltration in any part of the organ. Sections stained with methylene-blue show abundant bacilli, corresponding to those obtained in cultures, in the necrotic uterine lining. They have penetrated but a short distance into the tissues and are not found in the deeper layers of the mucosa, nor in the muscular or serous layers.

Lungs.—The lesions in the lungs are those of a hæmorrhagic pneumonia. The exudate is in part made up of small round cells (multinuclear). In other parts it is almost entirely hæmorrhagic, containing few or no leucocytes. In these latter areas the blood is, for the most part, well preserved, and some round cells contain pigment. When stained with methylene-blue and eosin, numerous bacilli are found in the hæmorrhagic areas. In places they are so numerous as to be easily recognized in mass with the low power. In the areas where the exudate consists of small round cells, few bacilli are to be found.

Liver.—Extensive fatty degeneration of the liver cells extends throughout the lobules.

Kidney.—The epithelium of the convoluted tubules is swollen and cloudy. The glomeruli are distended with blood, in some escaped blood being seen free in the capsules. There are a few small areas of hæmorrhage in the cortex.

Spleen.—There is a hyperplasia of the cells, and many large cells containing pigment.

DESCRIPTION OF ORGANISM.

Cultural Peculiarities.—In gelatine plates at room temperature, after 48 hours, the deep colonies appear to the naked eye as pin-point sized, white growths, and with a No. 3 Leitz objective, as round or slightly oval, pale yellowish, with an even outline and finely granular. After the same length of time the superficial colonies appear to the naked eye as about 1 mm. in diameter, glistening white, slightly elevated, with even or finely serrated edges. With the No. 3 Leitz objective they are translucent, with finely serrated edges, and uniformly and finely granular.

On agar-agar after 48 hours at 37° C. the superficial colonies were quite characteristic. The colony consisted of three zones. The centre was transparent or translucent white. About this was a zone of opaque white, while a third peripheral zone was transparent like the centre. The edges were finely irregular, and the colonies about $\frac{3}{16}$ inch in diameter.

The growth upon an agar-agar slant was abundant, white and porcelain-like.

On gelatine there was a fine granular growth along the line of puncture, and at the surface a fine growth which did not spread over the surface, nor become elevated.

On Loeffler's blood-serum mixture there was an abundant creamy, white growth, with gas production in the water of condensation. In glucose-agar there was abundant gas production. Bouillon was rendered diffusely cloudy. On potato there was a yellowish-white growth, elevated and with an irregular surface, which after a few days tended to extend over the surface beyond the site of inoculation.

Litmus-milk was turned faintly pink in 24 hours at 37° C., and in a week had lost some color, but there was no coagulation. There is no odor to the cultures, and no production of indol or phenol.

The growth is not so luxuriant when oxygen is excluded.

Growth is rapid at a temperature of 37° C., less so at 20° C. Slow growth occurs at as low as 7° C.

Morphology and Staining Properties.—The bacilli from cultures upon agar-agar, and blood serum, are non-motile. They stain readily with the aniline dyes. With carbol-fuchsin there is often a more intense staining at the poles, which is not present in specimens stained with Loeffler's methylene-blue. The bacilli are short, two to four times as long as thick, with rounded ends. They resemble the bacilli of chicken-cholera. They occur singly, often in pairs, but never in long strings. They are decolorized by Gram's method. No spore formation was observed.

Effects upon Animals.—Guinea-pigs and mice (house and white) died in from 15 to 36 hours after subcutaneous inoculations. The fatal dose of a 24-hour bouillon culture, at 37° C., was 0.5 cc. in guinea-pigs, and from 15 to 45 drops in mice.

In guinea-pigs, at the autopsy, there was found a reddish,

gelatinous exudate extending into the tissues to some distance from the point of inoculation. The adjacent lymph glands were swollen and very red. The serous cavities usually contained a blood-stained fluid exudate. The viscera of the abdomen and thorax did not show much macroscopic change except a swelling of the spleen, and perhaps an increased amount of blood in the lungs. Cultures from the blood in the heart, the various viscera, and serous cavities, always showed large numbers of bacilli everywhere. Sections of the various organs after hardening in alcohol allowed the bacilli to be demonstrated in the smaller vessels and capillaries. The tissue changes were not marked. There was fatty degeneration of the liver cells; distention of the capillaries in the glomerular loop in the kidney, with hæmorrhage into the capsule; areas of hæmorrhage in the spleen, and areas of hæmorrhagic pneumonia.

In mice there was at times some œdematous infiltration at the point of inoculation, but usually none. The internal organs were macroscopically little changed, except an enlargement of the spleen and a reddish mottling of the lungs. Smear preparations and cultures showed the bacilli to be present in all the organs and in the blood in the heart. In sections stained with Loeffler's methylene-blue the smaller vessels and capillaries of the various organs contained numerous bacilli. The tissue changes consisted in an increased amount of blood in the spleen, and fine areas of hæmorrhagic pneumonia in the lungs. In the pneumonic areas the bacilli were numerous.

Rabbits are almost entirely immune. After subcutaneous inoculations a local reaction occurs, but recovery follows. One young rabbit died after an injection of a very large quantity, probably from intoxication rather than from infection.

White rats and pigeons were entirely refractory.

This bacillus is an example of the bacteria classified by Hueppe as the cause of true hæmorrhagic septicæmia. They are usually short bacilli, which appear as diplococci because of the deeper end-staining. They do not spread over the surface of the gelatine, and usually do not grow on potato. They cause a true septicæmia, and invading the general vascular system cause a hæmorrhagic diathesis. A sharp line cannot be drawn between these and bacteria, which also cause multiple necrosis by growth in certain small areas.

This organism resembles some of those already described, but varies in its effects upon animals, and in other vital properties.

ANTITOXIC RELATION BETWEEN BEE POISON AND HONEY (?).

BY G. H. STOVER, M. D., *Denver, Colo.*

Miss M., aged 35, single, consulted me on September 9, 1895, on account of the rather unusual swelling of her right cheek following a bee-sting received some days before; the whole right side of the face was considerably swollen and she felt some constitutional symptoms.

After treatment for five days she recovered, and on her final visit made the interesting statement that, while in the past she had never been able to eat honey, indeed, was nauseated by the smell of it, even, since being stung she had developed a craving for it, and found that she could eat it with complete satisfaction and with no ill results.

Will some of the immunization experimenters throw light on this occurrence?

PROCEEDINGS OF SOCIETIES.

THE JOHNS HOPKINS HOSPITAL MEDICAL SOCIETY.

Broadbent's Sign.—DR. CAMAC.

It is with regret that I announce that the patient with adherent pericardium, who exhibited very strikingly the Broadbent Sign, is unable to be here to-night. As the phenomenon has been demonstrated to this Society at a former meeting I trust it will not be amiss to have reference to the sign without showing the case.

Broadbent* describes this sign of adherent pericardium as follows: ". . . Marked systolic retraction of some of the lower ribs on the lateral or posterior aspect of the thorax *may sometimes be seen.* This phenomenon is best seen when the patient is sitting up in a good light and the movements of the chest are carefully observed from a short distance off, first from the front and then from the lateral aspect. When a pulsatile movement is seen over the lowest part of the left side of the chest posteriorly, it may at first sight appear to be expansile. On a more careful scrutiny it will be found that there is a tug on the false ribs during the cardiac systole and a sharp rebound during diastole, which can be felt as well as seen when the hand is laid flat upon the chest-wall at the spot; *it is more marked when a deep inspiration is made;* it may be seen occasionally, not only on the left side, but also on the right, especially if the patient leans over to the left. Here, it is not possible that the heart can be directly fixed to the chest-wall at the points of retraction by pericardial adhesions, as the lung tissue intervenes; but the explanation seems to be the following: The heart is, by means of the pericardium, adherent not only to the central tendon of the diaphragm but probably also to a large area of the fleshy or muscular portion of the diaphragm, and, it may be, to the anterior thoracic wall as well; as it contracts it drags upwards and inwards the less resistant

*Adherent Pericardium. John F. H. Broadbent, M. D., M. R. C. P.

fleshy part of the diaphragm towards the central tendon or anterior chest-wall; hence the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and downwards. It will always be found in such cases that the retracted positions of the chest-wall correspond to the floating ribs or costal cartilages of the lower ribs at the points of attachment of the diaphragm. (Systolic recession of the left subcostal angle and epigastrium does not necessarily imply the presence of pericardial adhesions.)"

"The above is a most important diagnostic sign of adherent pericardium *when present*, and is quite distinct from recession of the lower ribs in inspiration."

Twice in the course of the description Broadbent infers that this is not a constant sign of adherent pericardium. He says at the beginning of the description that the sign "may sometimes be seen," and again at the conclusion that it is a most important sign "when present." It is to the explanation of this inconstancy that one's attention is drawn.

I have been unable to find in any work upon anatomy an accurate description as to the extent of pericardial attachment to the diaphragm. The general arrangement of the pericardium would suggest a hammock in which the heart is slung, attached above to the cervical fascia and below to the diaphragm, the diaphragmatic attachment involving largely the central tendon.

McClellan* gives the following description: The pericardium "is *intimately adherent* to the middle leaflet of the tendon about the opening for the inferior vena cava, and *more loosely connected* to the muscular part on the left side". . . . "These connections" (together with those of the deep cervical fascia already mentioned) "of the pericardium are of great interest, for *if the entire diaphragm descends* in respiration it must draw with it the heart sac, and therefore exert more or less strain upon the vessels at the base of the heart. The author inclines to the belief that *the central portion* or *tendon of the diaphragm does not descend, although the lateral muscular portions do.* On one occasion, after the excision of the sixth, seventh and eighth ribs on the right side, he was able to examine the upper surface of the diaphragm during the forced efforts of inspiration under ether, and on another, after the evacuation of the contents of an enormous abscess, involving the left lobe of the liver, he could easily introduce his hand into the abscess cavity and detect *the lateral upheaving of the diaphragm* and the rapid pulsation of the heart. In the latter case, during the straining of the patient in the act of vomiting, it was observed that the diaphragm descended and ascended with spasmodic contractions, *but only upon the sides*, there being apparently little if any change in the relations of its central tendon."

These observations would lead to the conclusion that the action of the diaphragm is like that of a flying bird, the central tendon being the motionless body, while the muscular portions would suggest the flapping wings.

Here, too, would seem to be the explanation of why, in extensively adherent pericardia, this sign may be absent. For if the pericarditis have involved that portion of the pericardium attached to the muscular diaphragm, which is constantly engaged in respiratory acts and attached to the false ribs, the tug on these ribs will be more pronounced than had it involved that portion attached to the central tendon, which is stationary and not engaged in respiratory acts. This explanation is further borne out by Broadbent's observation that the sign "is more marked when deep inspiration is made," the diaphragm being thus rendered more tense and consequently better suited to allow the heart to tug upon the ribs.

May we then say that those cases of adherent pericardium, in which Broadbent's Sign is absent, are such as have the least extent of involvement of that portion of the pericardium attached to the muscular diaphragm?

While this may appear anatomically correct it can only be conclusively proven by careful measurement of the diaphragmatic attachment of the pericardium in both healthy and adherent pericardia.

* Regional Anatomy. Vol. I, pp. 272-273, George McClellan, M. D.

Aortic Aneurysm.—Dr. Brown.

This case came to the hospital about a month ago complaining of pain in both sides of the back, especially the left, and the epigastrium. He came from healthy stock that did not indulge in alcohol to any extent, but his own personal history was different, as he had indulged largely in alcohol and had been exposed many times to venereal disease though he denied lues. He had been a hard worker. The present attack commenced nine months ago with a definite pain in the lower part of the back, at first rather slight and not enough to prevent the performance of his usual duties. Later it increased and at last became so intense that work was impossible; by this time the pain had radiated to the right side occasionally. He had no marked cough and very few other symptoms except the pain, but that was so great that sleep was difficult and it was necessary to maintain firm pressure to secure relief. He obtained this by standing up and pressing the epigastrium or the lumbar region against the edge of the table.

When he came into the hospital the physical examination showed a systolic and a diastolic murmur with marked pulsation in the left side of the chest which lifted up the sternum and was well marked in the axillary line. There was no point of expansile pulsation in this area. In examination of this area no tumor was seen, but one of the examiners described a tumor felt by ballottement, though all others were unable to feel it. Over the pulsating area both sounds of the heart were heard, but there was no diastolic shock. An examination of the arteries showed very marked sclerosis, and pulsation of the abdominal aorta was obtained with difficulty. The pain has lessened markedly since he came into the hospital.

Perhaps it will be of interest in connection with this case to say something of the etiology of the trouble. When we consider the etiology of aneurysm we have mainly to discuss the question of arterio-sclerosis. In this there are many factors, one of the most important being that of heredity. In this case that point applies but very slightly. Next comes the diffusion of certain poisons throughout the system, among which the most important are alcohol, and the poisons of gout, rheumatism and

syphilis. Exactly how these poisons act it is impossible to say definitely, but probably some at least act upon the adventitia of the arteries, and cause changes there which are finally compensated by hyperplasia of the intima. We simply have to take the etiology of arterio-sclerosis to work out the anatomical etiology of aneurysm. Given a man with weak arteries, and let him live a quiet life with little strain, he probably will not develop an aneurysm, but allow that man to do very hard work in which the strain upon the arterial system is very great and you have a great possibility of producing the disease.

In the question of diagnosis of aneurysm of the aorta there are various signs and symptoms according to the different portions affected. The aneurysms of the aorta may be divided into those of the ascending thoracic branch, of the arch, of the descending thoracic branch and of the abdominal aorta. In the first we have more signs than in any of the others. There is very marked pulsation, sometimes a tumor, though often few pressure symptoms, while, in the case of the transverse arch, the pressure symptoms are more marked, but the physical signs are less so. As we go into the descending portion of the thoracic aorta we may have more of the pressure symptoms, while as we proceed to the lower thoracic and upper portion of the abdominal aorta the symptoms become less because the chances of pressure are diminished. An aneurysm can be simulated by so many other conditions in the abdomen that Dr. Osler has said in his work that without the discovery of an expansile tumor, which can be grasped, a definite diagnosis cannot be made in many cases.

As regards the treatment, it is indeed multiform. Diet of a very dry character with rest in bed is given with the hope of diminishing the pulsation and increasing the formation of fibrinous elements in the blood. Various operative treatments, such as wiring of the sac and electrolysis, have been performed with the object of coagulating the fluid contents of the sac, with slight success, however, in most cases.

We attempted to clear up the diagnosis in this case by means of an X-ray photograph, but it was not much of a success. In looking up the subject of the application of X-ray photography to aneurysmal cases, however, I find that many have given it up, as better results are secured by use of the fluoroscope, carefully watching for a pulsating tumor. Whether the pulsating tumor will be shown by the fluoroscope in this case it is difficult to say, as fluoroscopic and radiographic work in the chest and abdomen are very difficult, but the signs and symptoms in this case are strongly in favor of its being a case of aneurysm, probably of the lower portion of the thoracic aorta.

Discussion of Mr. MacCallum's Paper on Pathology of Heart Muscle.—[See BULLETIN for Aug., p. 194.]

DR. FLEXNER: Mr. MacCallum's acquaintance with the embryology and histogenesis of the heart muscle made the pathology of the same muscle a peculiarly suitable subject of study. One must admire the technical method which resolves the muscle fibres into structures almost as complex as organs. Of much interest is the part played by these elementary constituents in the pathology of the fibre. In addition to the demonstration of the minuter changes in the fibres, the light shed on the nature of fragmentation of the heart muscle—whether due to irregularities in contraction or degeneration—is considerable. The necessary conclusion from these studies is that there is at least one form of fragmentation which cannot be regarded as agonal in origin—the one preceded by what Mr. MacCallun denominates sarcolytic degeneration. Whether in the light of this study we are to agree with the French writers who would make of this condition a disease *per se* is another question and one still to be answered.

The study of fibrous myocarditis must interest those who pay attention to histogenesis. The facts elicited are, to say the least, unexpected, and an explanation of the order of the degeneration does not seem apparent.

Epidemic Cerebro-Spinal Meningitis—Exhibition of Specimens.—DR. LIVINGOOD.

The present case is of interest as being one of several cases of epidemic spinal meningitis occurring in Baltimore which has come to autopsy. Some of you here present witnessed the autopsy. I do not think it necessary to go into minute details of the lesions generally met with in epidemic meningitis, as they have just been given in detail in Councilman, Mallory and Wright's monograph, which is accessible to you. I shall therefore confine my remarks to the case under consideration.

The patient was a boy aged 13 years. He was moderately well nourished; there was no external eruption; the skull-cap was of average thickness; the external (hard) meninges were injected; the vessels of the cortical pia were also injected, but there was no excess of fluid or any exudate in the cortical moderated. The exudate covered the basal portion of the brain, and existed in the form of a thick, adherent, creamy membrane, extending over pons, medulla and throughout the spinal canal. The ventricles were dilated, especially the lateral ventricles, and in the most dependent parts contained a turbid fluid. The walls of the ventricles were softened and somewhat macerated. The only other organs of interest besides the brain are the lungs. The right lung was free from adhesions; no pleurisy. The lower lobe was deeply congested, and contained four or five small areas of consolidation each the size of a marble. On section these were coarsely granular and variegated, pale or red, in color. The upper lobe contained about its mid-portion and near the pleura a consolidated focus the size of an orange. The pleura was congested over it and covered with a fibrinous membrane. The contained bronchi were dilated and contained purulent contents. On section it also was coarsely granular and variegated in appearance.

The bacteriological examination consisted in the study of cover-slips from the exudate in brain and cord and the consolidated foci in the lungs, and the examination of cultures from these several sources. The films made from the meningeal exudate showed only doubtful organisms; those made from the lungs showed many bacteria, chiefly within cells, which were in the form of diplococci, in many ways resembling the gonococcus, except for the absence of the buscuit-like flattening. In other cells the forms resembled more the lanceolate coccus. Tested by Gram's method most of the cocci gave up the dye; only those presenting lancet shapes seemed to retain it.

The cultures were interesting. Lumbar puncture made during life gave a positive result in that films and cultures

showed diplococci agreeing with the meningococcus intracellularis of Weichselbaum and Jaeger. At the autopsy cultures from the heart's blood, lungs, (consolidation) spleen, kidney, liver, brain and spinal meninges were made. Positive results were obtained only from the lung and meningeal exudate. The plate from the exudate over the medulla contained very few colonies of the meningococcus. The organisms isolated from the lung proved to be the micrococcus lanceolatus.

The meningococcus as obtained from the exudate presented the usual characteristics as given by Weichselbaum, Jaeger and Councilman, and did not retain the dye when treated by Gram's method. The culture from the lung failed in so far as the meningococcus was concerned, which was believed to be present there from the study of films, and which was shown by Councilman to be capable of setting up pneumonic conditions. As the subject of the nature and classification of these organisms has so recently been reviewed in the BULLETIN (J. H. H. BULLETIN No. 83), I shall not take up your time in restating the present views concerning them.

NOTES ON NEW BOOKS.

The Psychology of Suggestion. By BORIS SIDIS, M. A., Ph.D., Associate in Psychology at the Pathological Institute of the New York State Hospitals. With an Introduction by Prof. WILLIAM JAMES, of Harvard University. (*D. Appleton & Co., New York*, 1898)

Dr. Sidis, after devoting a chapter to the discussion of the meaning of the term "suggestion," all previous definitions of which he finds unsatisfactory, submits the following:

"By suggestion is meant the intrusion into the mind of an idea; met with more or less opposition by the person; accepted uncritically at last; and realized unreflectively, almost automatically."

Thus, if I ask a friend to lend me five dollars, it is a suggestion only in case he is reluctant to do so, and, nevertheless, finally gives me the money without thinking of his prospects of reimbursement. If he lends it to me willingly, or with the expectation of getting it back, or if he declines to let me have it at all, it is not a suggestion. This seems like a pretty arbitrary limitation, and it would be easy to show, by citations, that Dr. Sidis, in practice, dispenses with every one of the limitations which he imposes on the intruding idea.

It does not follow, because a writer fails in the definition of his subject that he may not have something of interest to say about it. Dr. Sidis has made many interesting observations, and presents the facts in regard to hypnotism and allied conditions, with which, naturally, the book is largely occupied, clearly and temperately. But, as might perhaps be expected from such a beginning, his inferences are not always warranted by the facts on which they are based.

The book is divided into three parts, the first treating of suggestibility, normal and abnormal, the second of the self, or personality, the third of the relations of suggestion to social phenomena.

In the first part the author undertakes to determine the laws of normal and abnormal suggestibility. The former he concludes to be as follows:

"Normal suggestibility varies as indirect suggestion, and inversely as direct suggestion."

In ordinary language, this means that if you want a normal person to do something, the surest way to do it is to conceal your wishes from him, while insinuating the idea of the action into his mind. So stated, it is evident at once that it is not a universal or even a general law. Dr. Sidis must be more unfortunate than most people in his acquaintances if their knowledge that he would like them to do so and so would not be some inducement to them to do it even uncritically and somewhat against their inclinations.

The law of abnormal suggestibility he formulates as follows:

"Abnormal suggestibility varies as direct suggestion, and inversely as indirect suggestion."

That is, in dealing with an abnormally suggestible person, the more explicit and emphatic the commands are made, the more likely they are to be executed. Dr. Sidis evidently has the hypnotic condition in mind, but it is by no means true that persons in this state are always insusceptible to indirect suggestion. As Dr. Sidis himself points out, the fallacy in the accounts given by Bernheim, Charcot and others of the various stages of the hypnotic state is due to the fact that the symptoms had been indirectly and unconsciously suggested to the patients by the operator. But hypnotism is not the only condition of abnormal suggestibility. In "negativism," or "contrariness," the patients may be led to do anything desired by telling them to do just the opposite.

The fact is, that in a normal condition, every suggestion, direct or indirect, tends to be carried out, but, on the other hand, it is apt to suggest, indirectly, conflicting ideas, which may inhibit it. In the hypnotic state, on the contrary, the conflicting considerations would seem to be largely or completely in abeyance.

In the second part, on "The Self," Dr. Sidis considers the nature of personality and the relations of what he calls the subconscious subwaking or secondary self, as manifested in such conditions as hypnotism, double consciousness and hysteria, to the primary or waking self. This is much the most interesting portion of the work. The account of the case of Rev. Thomas C. Hanna, who suffered complete loss of memory as the result of an accident, and of the means and steps by which the lost connection was recovered, is of remarkable interest, and the more detailed account which is promised will be most welcome to all readers of the book who are students of such subjects.

Perhaps the most original contribution of facts to be found in the book is the account of experiments tending to show that some degree of the abnormal acuteness of the senses which is a well-known phenomenon in many cases of hypnotism and hysteria is subconsciously present, to a certain extent in the normal condition. Thus, the author found that when words, letters or figures were shown at such a distance that they appeared as a mere confused blur, if the subjects of the experiments were required to guess what was shown them, their guesses were correct in a much larger proportion of cases than could be accounted for by chance. If his results in this direction are confirmed by subsequent investigators, they would seem to constitute a distinct advance in this department of psychology.

Although the tendency of the book is to show the subconscious presence, in normal conditions, of mental phenomena which are usually thought of as peculiar to abnormal states, Dr. Sidis does not seem fully to appreciate the fact that there is really no sharp distinction between our conscious and subconscious selves, but that they are constantly passing into and out of each other. Without attempting to criticise his discussion of the essential nature of personality, it may be said that our conception of our own personality in any given case is made up of our present sensations and feelings and our memories of the past. Only a small portion of all our experiences occupy our attention at any given moment; many can be readily called up; many more are lost beyond the possibility of recollection. We have entirely forgotten how we first learned to walk and to talk, for instance, although the knowledge then gained still abides with us. In the states of abnormal consciousness which the author has in mind, great blocks of the knowledge which is ordinarily at our command may be, for the time being, as much out of

reach as the recollection of the events of infancy. And just as we accept the account of others as to the events of times which have passed from memory, and incorporate them in our conception of our personality, so the hypnotic subject may accept the statements of the operator as to who and what he is, and govern himself accordingly.

As a physical basis for the phenomena of association and dissociation of states of consciousness, the author assumes the truth of the theory of contractility of the neuron. However convinced he may personally be of its correctness, it is hardly fair to his non-professional readers to give no hint of the fact that it is, thus far, a mere hypothesis, without, as far as at present appears, even the possibility of experimental verification, and open to very grave theoretical objections.

In the chapter on "Subconsciousness and Insanity," Dr. Sidis brings out clearly the analogy between post hypnotic suggestion and morbid impulses and imperative conception. Paranoia is much less satisfactorily treated.

In the third part of the book, treating of the psychology of crowds and mobs, and of "crazes" of various sorts, the mistake is made of confounding quite distinct phenomena. The influence exerted on susceptible persons by the presence of a multitude filled with a common emotion may very probably be, in some respects, analogous to the hypnotic condition, but there would, doubtless, have been a stampede for the Klondike if every one of those who went had supposed he had private and exclusive information of the riches to be gained there.

On the whole, the book, while containing much of interest, is valuable rather for its fact than its reasonings.

The Diseases of the Stomach. By WILLIAM W. VAN VALZAH, A. M., M. D., and J. DOUGLAS NISBET, A. B., M. D. Illustrated. (*Philadelphia: W. B. Saunders*, 1898.)

The stomach is a very important viscus, notwithstanding the fact that recent surgical successes have shown that under certain special conditions it can be dispensed with in the economy. Its importance is sufficiently in evidence when valuable text-books on its ailments follow each other with the rapidity which they have within the last two or three years. The present volume, while not supplanting the valuable treatises that have preceded it, has merits of its own, and is apparently well suited for a text-book on its subject.

The authors' plan of their work is a simple one, giving first the general methods of diagnosis and the general medications, then following this successively by sections on the dynamic affections of the stomach, including under this head all the symptomatic disorders that present, so far as known, no characteristic pathological anatomy, on the anatomical diseases which are, on the other hand, thus characterized, and finally ending with a section on the "vicous circles of the stomach," on the action of gastric disorders in producing or in being produced by disease elsewhere. This classification may be open to some criticism, but, on the whole, it seems fairly well adapted to afford a general view of the morbid conditions in which the stomach plays a chief or principal part. It is not especially original in its contents, and will hardly displace the recently published and excellent work of Hemmeter as a favorite with the American physician, but, as already said, it has its merits, and is well worthy of being an addition to any medical library.

Public Health Reports. (Formerly abstract of Sanitary Reports.) Issued by the Supervising Surgeon-General of the Marine Hospital Service. Vol. XII, Nos. 1–53. (*Washington: Government Printing Office*, 1898.)

This volume contains the reports of sanitary inspectors, United States consuls, and others on health conditions in foreign parts, translations from foreign languages of papers on special epidemics, and statistics of municipal health authorities here and abroad, of commissioners of emigration, etc., etc.; altogether a vast amount of valuable information upon sanitary matters. The work is a very useful one for reference in regard to these subjects, and the series of three volumes must form a very valuable record of sanitary statistics throughout the world, while the monthly publication of the parts that compose them is a useful current record.

The Archives of the Roentgen Rays. (Formerly Archives of Skiagraphy.) The only journal in which the transactions of the Roentgen Society of London are officially reported. Edited by W. S. HEDLEY, M. D., and SIDNEY ROWLAND, M. A. (*London: The Rebman Publishing Co.*, 1898.)

The title of this journal sufficiently indicates its nature and scope. It is elegantly printed and illustrated, and should be well received by those who use the Roentgen rays in physical or medical research. The only thing one can say against it is, that with the widening range of utility of Roentgen's discovery in surgery and medicine, a less expensive journal issued more frequently would be still more welcome to the medical profession. An actinoscopic adjunct is becoming almost essential to a surgeon's outfit, or at least such must be available to him, and there is every reason to believe that new utilities will be found, as time passes, for this method.

BOOKS RECEIVED.

Tenth Report of the State Board of Health of the State of Maine for the Two Years Ending Dec. 31, 1897. 395 pages. 8vo. 1898. Kennebec Journal Print, Augusta.

Practical Urinalysis and Urinary Diagnosis. A manual for the use of physicians, surgeons, and students. By Charles W. Purdy, M. D , LL. D. Fourth revised edition. 8vo. 1898. 365 pages. The F. A. Davis Co., Phila.

Essentials of Materia Medica, Therapeutics and Prescription Writing. Arranged in the form of questions and answers. Prepared especially for Students of Medicine. (Saunders' Question-Compends, No. 7.) By Henry Morris, M. D. 12mo. 1898. 288 pages. W. B. Saunders, Phila.

A Primer of Psychology and Mental Disease. For use in training-schools, for attendants and nurses and in medical classes. By C. B. Burr, M. D. Second edition, thoroughly revised. 12mo. 1898. 116 pages. The F. A. Davis Co., Phila.

The Care of the Baby. A manual for mothers and nurses, containing practical directions for the management of infancy and childhood in health and in disease. By J. P. Crozer Griffith, M. D. Second edition, revised. 8vo. 1898. 404 pages. W. B. Saunders, Phila.

Transactions of the Association of American Physicians. Thirteenth Session. Held at Washington, D. C., May 3, 4 and 5, 1898. Vol. XIII. 1898. 8vo, 484 pp. Printed for the Association. Phila.

Operative Gynecology. By Howard A. Kelly, A. B., M. D. Vol. II. 1898. 4to, 557 pp. D. Appleton & Co., New York.

Lehrbuch der Allgemeinen Pathologie und der pathologischen Anatomie. Von Dr. E. Ziegler. Zwei Bände. Neunte neu bearbeitete Auflage. Zweiter Band. Specielle pathologische Anatomie. 1898. 8vo, 1024 pp. Gustav Fischer, Jena.

The Pocket Formulary for the Treatment of Disease in Children. By Ludwig Freyberger, M. D., Vienna. 16 mo. 1898. 208 pages. The Rebman Publishing Co., Limited, London.

Transactions of the Michigan State Medical Society for the Year 1898. Volume XXII. 8vo. 1898. 450 pages. Published by the Society, Grand Rapids.

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GENERAL STATEMENT.

The Medical Department of the Johns Hopkins University was opened for the instruction of students October, 1893. This School of Medicine is an integral and coördinate part of the Johns Hopkins University, and it also derives great advantages from its close affiliation with the Johns Hopkins Hospital.

The required period of study for the degree of Doctor of Medicine is four years. The academic year begins on the first of October and ends the middle of June, with short recesses at Christmas and Easter.

Men and women are admitted upon the same terms.

In the methods of instruction especial emphasis is laid upon practical work in the Laboratories and in the Dispensary and Wards of the Hospital. While the aim of the School is primarily to train practitioners of medicine and surgery, it is recognized that the medical art should rest upon a suitable preliminary education and upon thorough training in the medical sciences. The first two years of the course are devoted mainly to practical work, combined with demonstrations, recitations and, when deemed necessary, lectures, in the Laboratories of Anatomy, Physiology, Physiological Chemistry, Pharmacology and Toxicology, Pathology and Bacteriology. During the last two years the student is given abundant opportunity for the personal study of cases of disease, his time being spent largely in the Hospital Wards and Dispensary and in the Clinical Laboratories. Especially advantageous for thorough clinical training are the arrangements by which the students, divided into groups, engage in practical work in the Dispensary, and throughout the fourth year serve as clinical clerks and surgical dressers in the wards of the Hospital.

REQUIREMENTS FOR ADMISSION.

As candidates for the degree of Doctor of Medicine the school receives:

1. Those who have satisfactorily completed the Chemical-Biological course which leads to the A. B. degree in this university.

2. Graduates of approved colleges or scientific schools who can furnish evidence: (*a*) That they have acquaintance with Latin and a good reading knowledge of French and German; (*b*) That they have such knowledge of physics, chemistry, and biology as is imparted by the regular minor courses given in these subjects in this university.

The phrase "a minor course," as here employed, means a course that requires a year for its completion. In physics, four class-room exercises and three hours a week in the laboratory are required; in chemistry and biology, four class-room exercises and five hours a week in the laboratory in each subject.

3. Those who give evidence by examination that they possess the general education implied by a degree in arts or in science from an approved college or scientific school, and the knowledge of French, German, Latin, physics, chemistry, and biology above indicated.

Applicants for admission will receive blanks to be filled out relating to their previous courses of study.

They are required to furnish certificates from officers of the colleges or scientific schools where they have studied, as to the courses pursued in physics, chemistry, and biology. If such certificates are satisfactory, no examination in these subjects will be required from those who possess a degree in arts or science from an approved college or scientific school.

Candidates who have not received a degree in arts or in science from an approved college or scientific school, will be required (1) to pass, at the beginning of the session in October, the matriculation examination for admission to the collegiate department of the Johns Hopkins University, (2) then to pass examinations equivalent to those taken by students completing the Chemical-Biological course which leads to the A. B. degree in this University, and (3) to furnish satisfactory certificates that they have had the requisite laboratory training as specified above. It is expected that only in very rare instances will applicants who do not possess a degree in arts or science be able to meet these requirements for admission.

Hearers and special workers, not candidates for a degree, will be received at the discretion of the Faculty.

ADMISSION TO ADVANCED STANDING.

Applicants for admission to advanced standing must furnish evidence (1) that the foregoing terms of admission as regards preliminary training have been fulfilled, (2) that courses equivalent in kind and amount to those given here, preceding that year of the course for admission to which application is made, have been satisfactorily completed, and (3) must pass examinations at the beginning of the session in October in all the subjects that have been already pursued by the class to which admission is sought. Certificates of standing elsewhere cannot be accepted in place of these examinations.

SPECIAL COURSES FOR GRADUATES IN MEDICINE.

Since the opening of the Johns Hopkins Hospital in 1889, courses of instruction have been offered to graduates in medicine. The attendance upon these courses has steadily increased with each succeeding year and indicates gratifying appreciation of the special advantages here afforded. With the completed organization of the Medical School, it was found necessary to give the courses intended especially for physicians at a later period of the academic year than that hitherto selected. It is, however, believed that the period now chosen for this purpose is more convenient for the majority of those desiring to take the courses than the former one. The special courses of instruction for graduates in medicine are now given annually during the months of May and June. During April there is a preliminary course in Normal Histology. These courses are in Pathology, Bacteriology, Clinical Microscopy, General Medicine, Surgery, Gynecology, Dermatology, Diseases of Children, Diseases of the Nervous System, Genito-Urinary Diseases, Laryngology and Rhinology, and Ophthalmology and Otology. The instruction is intended to meet the requirements of practitioners of medicine, and is almost wholly of a practical character. It includes laboratory coursee, demonstrations, bedside teaching, and clinical instruction in the wards, dispensary, amphitheatre, and operating rooms of the Hospital. These courses are open to those who have taken a medical degree and who give evidence satisfactory to the several instructors that they are prepared to profit by the opportunities here offered. The number of students who can be accommodated in some of the practical courses is necessarily limited. For these the places are assigned according to the date of application.

The Annual Announcement and Catalogue will be sent upon application. Inquiries should be addressed to the

REGISTRAR OF THE JOHNS HOPKINS MEDICAL SCHOOL, BALTIMORE.

The Johns Hopkins Hospital Bulletins are issued monthly. They are printed by THE FRIEDENWALD CO., Baltimore. Single copies may be procured from Messrs. CUSHING & CO. and the BALTIMORE NEWS COMPANY, Baltimore. Subscriptions, $1.00 a year, may be addressed to the publishers, THE JOHNS HOPKINS PRESS, BALTIMORE; single copies will be sent by mail for fifteen cents each.